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(CS) field
NEWS 4 AUG 24 ENCOMPLIT/ENCOMPLIT2 reloaded and enhanced
NEWS 5 AUG 24 CA/CAPplus enhanced with legal status information for
U.S. patents
NEWS 6 SEP 09 50 Millionth Unique Chemical Substance Recorded in
CAS REGISTRY
NEWS 7 SEP 11 WPIDS, WPINDEX, and WPIX now include Japanese FTERM
thesaurus
NEWS 8 OCT 21 Derwent World Patents Index Coverage of Indian and
Taiwanese Content Expanded
NEWS 9 OCT 21 Derwent World Patents Index enhanced with human
translated claims for Chinese Applications and
Utility Models

NEWS EXPRESS MAY 26 09 CURRENT WINDOWS VERSION IS V8.4,
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FILE 'HOME' ENTERED AT 14:53:10 ON 21 OCT 2009

=> file registry		
COST IN U.S. DOLLARS	SINCE FILE	TOTAL
	ENTRY	SESSION
FULL ESTIMATED COST	0.22	0.22

FILE 'REGISTRY' ENTERED AT 14:53:23 ON 21 OCT 2009
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DICTIONARY FILE UPDATES: 20 OCT 2009 HIGHEST RN 1189242-76-9

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L1 13 SURAMIN

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	5.83	6.05

FILE 'CAPLUS' ENTERED AT 14:53:45 ON 21 OCT 2009
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FILE COVERS 1907 - 21 Oct 2009 VOL 151 ISS 17
FILE LAST UPDATED: 20 Oct 2009 (20091020/ED)
REVISED CLASS FIELDS (/NCL) LAST RELOADED: Aug 2009
USPTO MANUAL OF CLASSIFICATIONS THESAURUS ISSUE DATE: Aug 2009

Caplus now includes complete International Patent Classification (IPC) reclassification data for the third quarter of 2009.

CAS Information Use Policies apply and are available at:

<http://www.cas.org/legal/infopolicy.html>

This file contains CAS Registry Numbers for easy and accurate substance identification.

=> s l1

L2 2137 L1

=> s 12 and (?cancer? or ?tumor? or ?tumour? or ?neoplasm?)

477220 ?CANCER?

750873 ?TUMOR?

6622 ?TUMOUR?

6622 ?TUMOUR?

751252 ?TUMOR?

(?TUMOR? OR ?TUMOUR?)

6622 ?TUMOUR?

750873 ?TUMOR?

750873 ?TUMOR?

751252 ?TUMOUR?

(?TUMOUR? OR ?TUMOR?)

583378 ?NEOPLASM?

L3 629 L2 AND (?CANCER? OR ?TUMOR? OR ?TUMOUR? OR ?NEOPLASM?)

=> s 13 and (cytotoxic? or chemothera?)

177175 CYTOTOXIC?

129850 CHEMOTHERA?

L4 205 L3 AND (CYTOTOXIC? OR CHEMOTHERA?)

=> s 14 and (?potentiat? or ?enhanc? or ?increas?)

91589 ?POTENTIAT?

1193058 ?ENHANC?

4865329 ?INCREAS?

L5 66 L4 AND (?POTENTIAT? OR ?ENHANC? OR ?INCREAS?)

=> s 15 and (kit or composition)

44707 KIT

42223 KITS

73613 KIT

(KIT OR KITS)

765279 COMPOSITION

353103 COMPOSITIONS

1110402 COMPOSITION

(COMPOSITION OR COMPOSITIONS)

L6 11 L5 AND (KIT OR COMPOSITION)

=> d 16 1-11 ibib abs

L6 ANSWER 1 OF 11 CAPLUS COPYRIGHT 2009 ACS on STN

ACCESSION NUMBER: 2008:493012 CAPLUS

DOCUMENT NUMBER: 148:509885

TITLE: Compositions and methods for treating neurological disorders or damage

INVENTOR(S): Diamandis, Phedias; Tyers, Mike; Dirks, Peter B.

PATENT ASSIGNEE(S): Can.

SOURCE: Can. Pat. Appl., 3pp.

CODEN: CPXXEB

DOCUMENT TYPE: Patent

LANGUAGE: English

FAMILY ACC. NUM. COUNT: 1

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
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CA 2606658	A1	20080413	CA 2007-2606658	20071012
US 20090076019	A1	20090319	US 2007-871562	20071012
PRIORITY APPLN. INFO.:			US 2006-851615P	P 20061013

AB The invention relates to a clonogenic neurosphere assay to carry out high throughput screens (HTS) to identify potent and/or selective modulators of

proliferation, differentiation and/or renewal of neural precursor cells, neural progenitor cells and/or self-renewing and multipotent neural stem cells (NSCs). The invention also relates to compns. comprising the identified modulators and methods of using the modulators and compns., in particular to treat neurol. disorders (e.g. brain or CNS cancer) or damage.

OS.CITING REF COUNT: 1 THERE ARE 1 CAPLUS RECORDS THAT CITE THIS RECORD
(1 CITINGS)

L6 ANSWER 2 OF 11 CAPLUS COPYRIGHT 2009 ACS on STN

ACCESSION NUMBER: 2006:586464 CAPLUS

DOCUMENT NUMBER: 145:130745

TITLE: Pharmaceutical composition containing plant alkaloids for treating solid tumor

INVENTOR(S): Kong, Qingzhong; Sun, Juan; Kong, Qingxin; Sun, Zhonghou

PATENT ASSIGNEE(S): Shandong Lanjin Biotech Co., Ltd., Peop. Rep. China

SOURCE: Faming Zhuanli Shenqing Gongkai Shuomingshu, 17 pp.

CODEN: CNXXEV

DOCUMENT TYPE: Patent

LANGUAGE: Chinese

FAMILY ACC. NUM. COUNT: 1

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
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CN 1686544	A	20051026	CN 2005-10042259	20050406

PRIORITY APPLN. INFO.: CN 2005-10042259 20050406

AB The title composition contains plant alkaloids and potentiators as active components and biocompatible and biodegradable polymers as auxiliary agents. The potentiators can be selected from platinum compds., tetrazine compds., and/or topoisomerase inhibitors. The topical sustained-release of effective components can reduce systemic toxic reaction, selectively increase the drug level at the tumor site, and improve the therapeutic effect of non-operative therapy such as chemotherapy and radiotherapy.

L6 ANSWER 3 OF 11 CAPLUS COPYRIGHT 2009 ACS on STN

ACCESSION NUMBER: 2006:529026 CAPLUS

DOCUMENT NUMBER: 145:110264

TITLE: Antitumor composition of topoisomerase inhibitor and guanine or guanine analogs

INVENTOR(S): Kong, Qingzhong; Sun, Juan; Kong, Qingxin; Su, Hongqing; Sun, Jing

PATENT ASSIGNEE(S): Shandong Lanjin Biotech Co., Ltd., Peop. Rep. China

SOURCE: Faming Zhuanli Shenqing Gongkai Shuomingshu, 19 pp.

CODEN: CNXXEV

DOCUMENT TYPE: Patent

LANGUAGE: Chinese

FAMILY ACC. NUM. COUNT: 1

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
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CN 1679945	A	20051012	CN 2005-10042429	20050203
CN 100402091	C	20080716		

PRIORITY APPLN. INFO.: CN 2005-10042429 20050203

AB The title antitumor composition comprises topoisomerase inhibitors, guanine or guanine analogs as effective components, and auxiliary materials. The guanine and its analogs can inhibit DNA repair in cells and decrease tumor cell tolerance to tetrazines drugs. The auxiliary materials are biocompatible and biodegradable polymers for

topical sustained-release of effective components. The topical release-release of effective components can reduce systemic toxic reaction, selectively increase the drug level at the tumor site, and improve the therapeutic effect of non-operative therapy such as chemotherapy and radiotherapy.

L6 ANSWER 4 OF 11 CAPLUS COPYRIGHT 2009 ACS on STN

ACCESSION NUMBER: 2006:491764 CAPLUS
DOCUMENT NUMBER: 145:1047
TITLE: Methods and compositions using sirtuin modulators for treating or preventing obesity and insulin resistance disorders
INVENTOR(S): Sinclair, David A.; Alexander-Bridges, Maria
PATENT ASSIGNEE(S): President and Fellows of Harvard College, USA; The General Hospital Corporation
SOURCE: U.S. Pat. Appl. Publ., 154 pp., Cont.-in-part of U.S. Ser. No. 27,779.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 2
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 20060111435	A1	20060525	US 2005-174000	20050701
US 20050171027	A1	20050804	US 2004-27779	20041229
AU 2006266125	A1	20070111	AU 2006-266125	20060628
CA 2613636	A1	20070111	CA 2006-2613636	20060628
WO 2007005453	A2	20070111	WO 2006-US25138	20060628
WO 2007005453	A3	20070614		
W:	AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HN, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KN, KP, KR, KZ, LA, LC, LK, LR, LS, LT, LU, LV, LY, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RS, RU, SC, SD, SE, SG, SK, SL, SM, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, ZA, ZM, ZW			
RW:	AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AP, EA, EP, OA			
EP 1912632	A2	20080423	EP 2006-774176	20060628
R:	AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LI, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR			
JP 2009500331	T	20090108	JP 2008-519513	20060628
PRIORITY APPLN. INFO.:			US 2003-533712P	P 20031229
			US 2004-588643P	P 20040716
			US 2004-27779	A2 20041229
			US 2005-174000	A 20050701
			WO 2006-US25138	W 20060628

AB The invention provides methods and compns. for modulating the activity or level of a sirtuin, thereby treating or preventing obesity or an insulin resistance disorder, e.g. diabetes, in a subject. Exemplary methods comprise contacting a cell with a sirtuin activating compound or an inhibitory compound to thereby increase or decrease fat accumulation, resp.

OS.CITING REF COUNT: 5 THERE ARE 5 CAPLUS RECORDS THAT CITE THIS RECORD (7 CITINGS)

L6 ANSWER 5 OF 11 CAPLUS COPYRIGHT 2009 ACS on STN

ACCESSION NUMBER: 2006:469720 CAPLUS
 DOCUMENT NUMBER: 144:460881
 TITLE: Methods and compositions for increasing stem cell homing using Gas activators
 INVENTOR(S): Scadden, David T.; Kronenberg, Henry; Adams, Gregor
 PATENT ASSIGNEE(S): The General Hospital Corporation, USA
 SOURCE: PCT Int. Appl., 36 pp.
 CODEN: PIXXD2
 DOCUMENT TYPE: Patent
 LANGUAGE: English
 FAMILY ACC. NUM. COUNT: 1
 PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2006052960	A2	20060518	WO 2005-US40416	20051107
WO 2006052960	A3	20071122		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KN, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, LY, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SM, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AP, EA, EP, OA				
US 20080112933	A1	20080515	US 2007-667329	20071102
PRIORITY APPLN. INFO.:			US 2004-625914P	P 20041108
			WO 2005-US40416	W 20051107

AB The invention provides methods for increasing engraftment of stem cells in a subject by treating the cells with a Gas activator. The invention further provides methods for identifying Gas activators for use in increasing engraftment of stem cells in a subject.

L6 ANSWER 6 OF 11 CAPLUS COPYRIGHT 2009 ACS on STN

ACCESSION NUMBER: 2006:372182 CAPLUS
 DOCUMENT NUMBER: 144:495317
 TITLE: Anticancer implantation composition containing angiogenesis inhibitor and antitumor agent
 INVENTOR(S): Kong, Qingzhong; Sun, Juan; Yu, Jianjiang
 PATENT ASSIGNEE(S): Peop. Rep. China
 SOURCE: Faming Zhuanli Shenqing Gongkai Shuomingshu, 19 pp.
 CODEN: CNXXEV
 DOCUMENT TYPE: Patent
 LANGUAGE: Chinese
 FAMILY ACC. NUM. COUNT: 1
 PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
CN 1733302	A	20060215	CN 2005-10044379	20050805
PRIORITY APPLN. INFO.:			CN 2005-10044379	20050805

AB The title anticancer implantation composition comprises an angiogenesis inhibitor, an antitumor agent (plant alkaloids, platinum compds., tetrazines, and/or topoisomerase inhibitors), and pharmaceutical auxiliary materials. The auxiliary materials are

biocompatible and degradable polymer which can slowly release the anticancer medicines at the tumor site during the degradation and absorption process. This composition can be placed at the tumor site to reduce systemic toxic reaction of the drugs, to increase the drug concentration selectively at the tumor site, and to improve the therapeutic effect of non-operative therapy, such as chemotherapy and radiotherapy.

L6 ANSWER 7 OF 11 CAPLUS COPYRIGHT 2009 ACS on STN

ACCESSION NUMBER: 2005:1257943 CAPLUS
DOCUMENT NUMBER: 144:135174
TITLE: Manufacture of anticancer medicinal composition containing topoisomerase inhibitors
INVENTOR(S): Kong, Qingzhong
PATENT ASSIGNEE(S): Peop. Rep. China
SOURCE: Faming Zhuanli Shenqing Gongkai Shuomingshu, 23 pp.
CODEN: CNXXEV
DOCUMENT TYPE: Patent
LANGUAGE: Chinese
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
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CN 1616099	A	20050518	CN 2004-10035927	20041014
CN 1299773	C	20070214		

PRIORITY APPLN. INFO.: CN 2004-10035927 20041014

AB The title composition contains nitrosoarea anticancer drugs (0.00-40 weight%) and topoisomerase inhibitors (0.01-50 weight%) enveloped in the medicinal adjuvant. Topoisomerase inhibitors can inhibit DNA repair in cells, and reduce the tolerance of tumor cells to nitrosoarea anticancer drugs. The medicinal adjuvant is biocompatible and degradable polymer, which can slowly release the anticancer active components at the tumor site during the degradation and absorption process so as to reduce the systemic toxic reaction while maintaining effective levels of the drugs at the tumor site. The composition can be placed at the tumor site to reduce systemic toxic reaction of nitrosoarea anticancer drugs and topoisomerase inhibitor, and also selectively increase the drug level at the tumor site so as to improve the therapeutic effect of non-operative therapy such as chemotherapy and radiotherapy.

L6 ANSWER 8 OF 11 CAPLUS COPYRIGHT 2009 ACS on STN

ACCESSION NUMBER: 2004:902199 CAPLUS
DOCUMENT NUMBER: 141:374704
TITLE: Composition and uses of galectin antagonists to augment treatment of cancer or other proliferative disorders
INVENTOR(S): Chang, Yan; Sasak, Vodek
PATENT ASSIGNEE(S): Glycogenesys, Inc., USA
SOURCE: PCT Int. Appl., 51 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 3
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
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WO 2004091634	A1	20041028	WO 2004-US10675	20040407
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH,				

CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD,
 GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC,
 LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI,
 NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY,
 TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
 RW: BW, GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ,
 BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE,
 ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT, RO, SE, SI,
 SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN,
 TD, TG
 US 20040023925 A1 20040205 US 2003-408723 20030407
 AU 2004229399 A1 20041028 AU 2004-229399 20040407
 CA 2521649 A1 20041028 CA 2004-2521649 20040407
 US 20040223971 A1 20041111 US 2004-819901 20040407
 EP 1617849 A1 20060125 EP 2004-759200 20040407
 EP 1617849 B1 20080618
 R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT,
 IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, PL, SK, HR
 JP 2006522163 T 20060928 JP 2006-509773 20040407
 IN 2005DN05019 A 20091002 IN 2005-DN5019 20051103
 US 20080089959 A1 20080417 US 2007-803150 20070511
 PRIORITY APPLN. INFO.:
 US 2003-408723 A 20030407
 US 2003-461006P P 20030407
 US 2003-474562P P 20030530
 US 2001-299991P P 20010621
 US 2002-176235 A2 20020620
 US 2004-819901 B1 20040407
 WO 2004-US10675 W 20040407
 AB The present invention is directed to methods and compns. for augmenting
 treatment of cancers and other proliferative disorders. In
 particular embodiments, the invention combines the administration of an
 agent that inhibits the anti-apoptotic activity of galectin-3 (e.g., a
 'galectin-3 inhibitor') so as to potentiate the toxicity of a
 chemotherapeutic agent. In certain preferred embodiments, the
 conjoint therapies of the present invention can be used to improve the
 efficacy of those chemotherapeutic agents whose
 cytotoxicity is influenced by the status of an anti-apoptotic
 Bcl-2 protein for the treated cell. For instance, galectin-3 inhibitors
 can be administered in combination with a chemotherapeutic agent
 that interferes with DNA replication fidelity or cell-cycle progression of
 cells undergoing unwanted proliferation.
 OS.CITING REF COUNT: 2 THERE ARE 2 CAPLUS RECORDS THAT CITE THIS RECORD
 (2 CITINGS)
 REFERENCE COUNT: 4 THERE ARE 4 CITED REFERENCES AVAILABLE FOR THIS
 RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT
 L6 ANSWER 9 OF 11 CAPLUS COPYRIGHT 2009 ACS on STN
 ACCESSION NUMBER: 2004:453016 CAPLUS
 DOCUMENT NUMBER: 141:1227
 TITLE: Combination cancer therapy with a
 glutathione S-transferase (GST)-activated
 anticancer compound and another
 anticancer therapy
 INVENTOR(S): Xu, Hua; Brown, Gail L.; Schow, Steven R.; Keck, James
 G.
 PATENT ASSIGNEE(S): Telik, Inc., USA
 SOURCE: PCT Int. Appl., 38 pp.
 CODEN: PIXXD2
 DOCUMENT TYPE: Patent
 LANGUAGE: English
 FAMILY ACC. NUM. COUNT: 1
 PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
-----	----	-----	-----	-----
WO 2004045593	A2	20040603	WO 2003-US36209	20031114
WO 2004045593	A3	20040812		
W:	AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, UZ, VC, VN, YU, ZA, ZM, ZW			
RW:	BW, GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG			
CA 2505377	A1	20040603	CA 2003-2505377	20031114
AU 2003290805	A1	20040615	AU 2003-290805	20031114
US 20040138140	A1	20040715	US 2003-714593	20031114
EP 1562564	A2	20050817	EP 2003-783388	20031114
R:	AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, SK			
BR 2003016364	A	20051004	BR 2003-16364	20031114
CN 1711076	A	20051221	CN 2003-80103404	20031114
CN 100508961	C	20090708		
JP 2006508980	T	20060316	JP 2004-553614	20031114
IN 2005CN00892	A	20070824	IN 2005-CN892	20050512
MX 2005005200	A	20050818	MX 2005-5200	20050513
US 20080159980	A1	20080703	US 2007-872659	20071015
PRIORITY APPLN. INFO.:			US 2002-426983P	P 20021115
			US 2003-714593	B1 20031114
			WO 2003-US36209	W 20031114

OTHER SOURCE(S): MARPAT 141:1227

AB The invention discloses a method for combination cancer therapy in a mammal, especially a human, by administering a therapeutically effective amount of a GST-activated anticancer compound and a therapeutically ED of another anticancer therapy. Also disclosed are pharmaceutical compns., products, and kits for the method, as well as the use of a GST-activated anticancer compound in the manufacture of a medicament for the method. The invention further discloses a method for potentiating an anticancer therapy in a mammal, especially a human, comprising administering a therapeutically effective amount of a GST-activated anticancer compound to the mammal being treated with the anticancer therapy. Further disclosed is the use of a GST-activated anticancer compound in the manufacture of a medicament for the method. The GST-activated anticancer compound is preferably a compound of US Patent Number 5,556,942, and more preferably TLK286, especially as the hydrochloride salt.

OS.CITING REF COUNT: 2 THERE ARE 2 CAPLUS RECORDS THAT CITE THIS RECORD (2 CITINGS)

REFERENCE COUNT: 2 THERE ARE 2 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L6 ANSWER 10 OF 11 CAPLUS COPYRIGHT 2009 ACS on STN

ACCESSION NUMBER: 2003:609872 CAPLUS

DOCUMENT NUMBER: 139:154909

TITLE: Compositions for delivery of antitumor drug combinations

INVENTOR(S): Tardi, Paul; Harasym, Troy; Webb, Murray; Shew, Clifford

PATENT ASSIGNEE(S): Can.

SOURCE: U.S. Pat. Appl. Publ., 67 pp.

DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 7
PATENT INFORMATION:

CODEN: USXXCO

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 20030147945	A1	20030807	US 2002-264538	20021003
CA 2383259	A1	20031023	CA 2002-2383259	20020423
EP 1693052	A1	20060823	EP 2006-9230	20021003
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI, CY, TR, BG, CZ, EE, SK				
AT 345775	T	20061215	AT 2002-766997	20021003
ES 2272768	T3	20070501	ES 2002-766997	20021003
US 20040022817	A1	20040205	US 2003-417631	20030416
US 20060193904	A1	20060831	US 2005-256445	20051021
US 20060165771	A1	20060727	US 2005-304328	20051214
US 20070148255	A1	20070628	US 2007-701326	20070131
US 20080044464	A1	20080221	US 2007-842130	20070820
US 20080075762	A1	20080327	US 2007-841786	20070820
AU 2007237323	A1	20071220	AU 2007-237323	20071204
AU 2007237323	B2	20090827		

PRIORITY APPLN. INFO.:
US 2001-326671P P 20011003
US 2001-341529P P 20011217
US 2002-356759P P 20020215
CA 2002-2383259 A 20020423
US 2002-401984P P 20020807
US 2002-408733P P 20020906
US 2002-362074P P 20020307
US 2002-394273P P 20020709
AU 2002-331480 A3 20021003
EP 2002-766997 A3 20021003
US 2002-264538 A2 20021003
US 2002-264818 A3 20021003
US 2003-406913 B1 20030402
US 2003-417631 A3 20030416
US 2005-256445 A1 20051021

AB Compns. which comprise delivery vehicles having stably associated therewith non-antagonistic combinations of two or more agents, such as antineoplastic agents, are useful in achieving non-antagonistic effects when combinations of drugs are administered. Thus, liposomal carboplatin and daunorubicin encapsulated at a 10:1 non-antagonistic mole ratio in sphingomyelin-containing liposomes exhibited substantially increased efficacy in relation to controls consisting of free drug and saline.

OS.CITING REF COUNT: 3 THERE ARE 3 CAPLUS RECORDS THAT CITE THIS RECORD (3 CITINGS)

L6 ANSWER 11 OF 11 CAPLUS COPYRIGHT 2009 ACS on STN

ACCESSION NUMBER: 1999:783929 CAPLUS

DOCUMENT NUMBER: 132:18780

TITLE: Compositions comprising antimicrotubule agents for treating or preventing inflammatory diseases

INVENTOR(S): Hunter, William L.

PATENT ASSIGNEE(S): Angiotech Pharmaceuticals, Inc., Can.

SOURCE: PCT Int. Appl., 340 pp.

CODEN: PIXXD2

DOCUMENT TYPE: Patent

LANGUAGE: English

FAMILY ACC. NUM. COUNT: 3

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9962510	A2	19991209	WO 1999-CA464	19990601
W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, GM, HR, HU, ID, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZA, ZW				
RW: GH, GM, KE, LS, MW, SD, SL, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
US 6495579	B1	20021217	US 1998-88546	19980601
AU 2006220416	A1	20061026	AU 2006-220416	20060920
AU 2006220416	B2	20090205		
PRIORITY APPLN. INFO.:			US 1998-88546	A 19980601
			US 1996-32215P	P 19961202
			US 1997-63087P	P 19971024
			US 1997-980549	A2 19971201
			AU 2004-200715	A3 20040220
AB Methods and compns. for treating or preventing inflammatory diseases, e.g. psoriasis or multiple sclerosis, are provided, comprising the step of delivering to the site of inflammation an antimicrotubule agent, or analog or derivative thereof.				
OS.CITING REF COUNT:		8	THERE ARE 8 CAPLUS RECORDS THAT CITE THIS RECORD (8 CITINGS)	
REFERENCE COUNT:		4	THERE ARE 4 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT	

=> d his

(FILE 'HOME' ENTERED AT 14:53:10 ON 21 OCT 2009)

FILE 'REGISTRY' ENTERED AT 14:53:23 ON 21 OCT 2009

L1 13 S SURAMIN

FILE 'CAPLUS' ENTERED AT 14:53:45 ON 21 OCT 2009

L2 2137 S L1
L3 629 S L2 AND (?CANCER? OR ?TUMOR? OR ?TUMOUR? OR ?NEOPLASM?)
L4 205 S L3 AND (CYTOTOXIC? OR CHEMOTHERA?)
L5 66 S L4 AND (?POTENTIAT? OR ?ENHANC? OR ?INCREAS?)
L6 11 S L5 AND (KIT OR COMPOSITION)

=> file registry

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	60.14	66.19
DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)	SINCE FILE ENTRY	TOTAL SESSION
CA SUBSCRIBER PRICE	-9.02	-9.02

FILE 'REGISTRY' ENTERED AT 14:56:51 ON 21 OCT 2009

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STRUCTURE FILE UPDATES: 20 OCT 2009 HIGHEST RN 1189242-76-9

DICTIONARY FILE UPDATES: 20 OCT 2009 HIGHEST RN 1189242-76-9

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TSCA INFORMATION NOW CURRENT THROUGH June 26, 2009.

Please note that search-term pricing does apply when
conducting SmartSELECT searches.

REGISTRY includes numerically searchable data for experimental and
predicted properties as well as tags indicating availability of
experimental property data in the original document. For information
on property searching in REGISTRY, refer to:

<http://www.cas.org/support/stngen/stndoc/properties.html>

=> s suramin/cn

L7 1 SURAMIN/CN

=> d 17

L7 ANSWER 1 OF 1 REGISTRY COPYRIGHT 2009 ACS on STN

RN 145-63-1 REGISTRY

ED Entered STN: 16 Nov 1984

CN 1,3,5-Naphthalenetrisulfonic acid,
8,8'-[carbonylbis[imino-3,1-phenylenecarbonylimino(4-methyl-3,1-
phenylene)carbonylimino]]bis- (CA INDEX NAME)

OTHER CA INDEX NAMES:

CN 1,3,5-Naphthalenetrisulfonic acid,
8,8'-[ureylenebis[m-phenylenecarbonylimino(4-methyl-m-
phenylene)carbonylimino]]di- (8CI)

OTHER NAMES:

CN 8,8'-[Ureylenebis[m-phenylenecarbonylimino(4-methyl-m-
phenylene)carbonylimino]]di-1,3,5-naphthalenetrisulfonic acid

CN Farma

CN Farma 939

CN Fourneau

CN Metaret

CN Naganol

CN Suramin

CN Suramine

MF C51 H40 N6 O23 S6

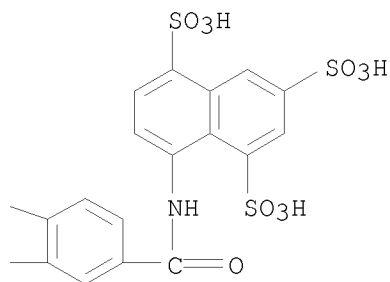
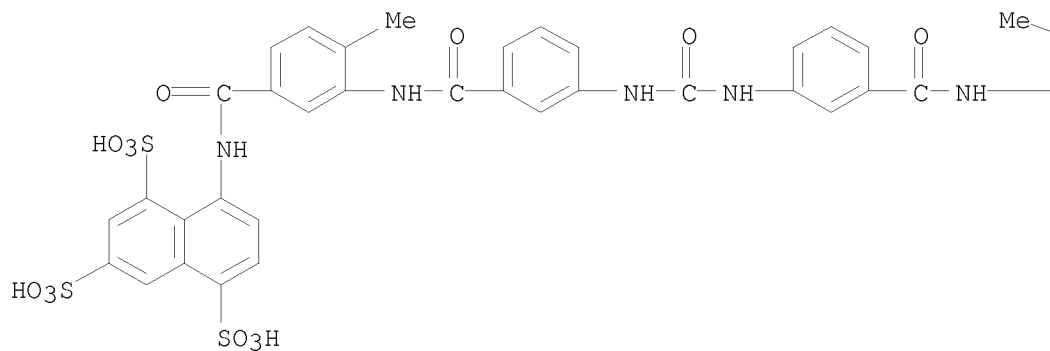
CI COM

LC STN Files: ADISINSIGHT, ADISNEWS, AGRICOLA, ANABSTR, AQUIRE, BEILSTEIN*,
BIOSIS, BIOTECHNO, CA, CABA, CAPLUS, CBNB, CHEMCATS, CHEMLIST, CIN,
CSCHEM, DDFU, DRUGU, EMBASE, IMSDRUGNEWS, IMSRESEARCH, IPA, MEDLINE,
NAPRALERT, PHAR, PROMT, PROUSDDR, RTECS*, SYNTHLINE, TOXCENTER, TULSA,
USAN, USPAT2, USPATFULL, USPATOLD, VETU

(*File contains numerically searchable property data)

Other Sources: EINECS**

(**Enter CHEMLIST File for up-to-date regulatory information)



PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

1779 REFERENCES IN FILE CA (1907 TO DATE)
 62 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
 1782 REFERENCES IN FILE CAPLUS (1907 TO DATE)

=> file caplus
 COST IN U.S. DOLLARS
 FULL ESTIMATED COST

SINCE FILE ENTRY	TOTAL SESSION
7.88	74.07

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)
 CA SUBSCRIBER PRICE

SINCE FILE ENTRY	TOTAL SESSION
0.00	-9.02

FILE 'CAPLUS' ENTERED AT 14:57:15 ON 21 OCT 2009
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FILE COVERS 1907 - 21 Oct 2009 VOL 151 ISS 17
FILE LAST UPDATED: 20 Oct 2009 (20091020/ED)
REVISED CLASS FIELDS (/NCL) LAST RELOADED: Aug 2009
USPTO MANUAL OF CLASSIFICATIONS THESAURUS ISSUE DATE: Aug 2009

CAPLUS now includes complete International Patent Classification (IPC) reclassification data for the third quarter of 2009.

CAS Information Use Policies apply and are available at:

<http://www.cas.org/legal/infopolicy.html>

This file contains CAS Registry Numbers for easy and accurate substance identification.

=> s 17

L8 1782 L7

=> s 18 and (?cancer? or ?tumor? or ?tumour? or ?neoplasm?)

477220 ?CANCER?

750873 ?TUMOR?

6622 ?TUMOUR?

6622 ?TUMOUR?

751252 ?TUMOR?

(?TUMOR? OR ?TUMOUR?)

6622 ?TUMOUR?

750873 ?TUMOR?

750873 ?TUMOR?

751252 ?TUMOUR?

(?TUMOUR? OR ?TUMOR?)

583378 ?NEOPLASM?

L9 605 L8 AND (?CANCER? OR ?TUMOR? OR ?TUMOUR? OR ?NEOPLASM?)

=> s 19 and (cytotoxic? or chemothera?)

177175 CYTOTOXIC?

129850 CHEMOTHERA?

L10 197 L9 AND (CYTOTOXIC? OR CHEMOTHERA?)

=> s 110 and ad<20010924

4146552 AD<20010924

(AD<20010924)

L11 21 L10 AND AD<20010924

=> d 111 1-21 ibib abs

L11 ANSWER 1 OF 21 CAPLUS COPYRIGHT 2009 ACS on STN

ACCESSION NUMBER: 2009:876469 CAPLUS

DOCUMENT NUMBER: 151:164294

TITLE: Gossypolone for the treatment of cancer

INVENTOR(S): Flack, Mary R.; Knazek, Richard; Reidenberg, Marcus

PATENT ASSIGNEE(S): The United States of America as Represented by the
Department of Health and Human Services, USA

SOURCE: U. S. Reissue, 10pp.
CODEN: UUXXA2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 2
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 40862	E1	20090721	US 2006-581734	20061016
US 551353	A0	19910415	US 1990-551353	19900712 <--
US 5385936	A	19950131		
US 6114397	A	20000905	US 1995-379872	19950127 <--
PRIORITY APPLN. INFO.:			US 1990-551353	A3 19900712
			US 1995-379872	E 19950127
			US 2004-806088	A1 20040322

AB The invention discloses a method for treating cancer in a human, which comprises administering an anti-cancer effective amount of a compound selected from gossypol, gossypol acetic acid, gossypolone, metabolites thereof, or physiol. acceptable salts thereof. Also included is a method for treating cancer in a human which comprises administering to the human subject an anti-cancer effective amount of any of the compds. listed above in combination with an anti-cancer effective amount of other conventional chemotherapeutic agents. Finally, the invention also encompasses a pharmaceutical composition comprising an anti-cancer effective amount of gossypol, gossypol acetic acid, or gossypolone, and an anti-cancer effective amount of a conventional chemotherapeutic agent, or combinations of the latter.

OS.CITING REF COUNT: 7 THERE ARE 7 CAPLUS RECORDS THAT CITE THIS RECORD
(7 CITINGS)

L11 ANSWER 2 OF 21 CAPLUS COPYRIGHT 2009 ACS on STN

ACCESSION NUMBER: 2005:1004423 CAPLUS
DOCUMENT NUMBER: 143:312080
TITLE: Artificial blood vessel for delivering therapeutic agents
INVENTOR(S): Bhat, Vinayak D.; Yan, John
PATENT ASSIGNEE(S): Avantec Vascular Corp., USA
SOURCE: U.S. Pat. Appl. Publ., 52 pp., Cont.-in-part of U.S. Ser. No. 206,807.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 2
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 20050203612	A1	20050915	US 2003-607836	20030627
US 20020082677	A1	20020627	US 2001-782804	20010213 <--
US 7018405	B2	20060328		
US 20020114823	A1	20020822	US 2001-782927	20010213 <--
US 6471980	B2	20021029		
US 20020082679	A1	20020627	US 2001-2595	20011101
US 20030083646	A1	20030501	US 2001-17500	20011214
US 7077859	B2	20060718		
US 20030050692	A1	20030313	US 2002-206807	20020725
US 20030017190	A1	20030123	US 2002-242334	20020911
US 6858221	B2	20050222		
WO 2004010900	A1	20040205	WO 2003-US20492	20030627

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN,

CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH,
 GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR,
 LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI, NO, NZ, OM,
 PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN,
 TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
 RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY,
 KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES,
 FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR,
 BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

AU 2003261100 A1 20040216 AU 2003-261100 20030627
 JP 2005533604 T 20051110 JP 2004-524538 20030627
 US 20070142898 A1 20070621 US 2007-680439 20070228
 PRIORITY APPLN. INFO.: US 2000-258024P P 20001222

US 2001-782804 A2 20010213
 US 2001-782927 A2 20010213
 US 2001-783253 A2 20010213
 US 2001-783254 A2 20010213
 US 2001-308381P P 20010726
 US 2001-2595 A2 20011101
 US 2001-17500 A2 20011214
 US 2002-347473P P 20020110
 US 2002-355317P P 20020207
 US 2002-370703P P 20020406
 US 2002-206807 A2 20020725
 US 2002-404624P P 20020819
 US 2003-454146P P 20030311
 US 2003-472536P P 20030521
 WO 2003-US20492 W 20030627

AB Devices and methods for reducing, inhibiting, or treating restenosis and hyperplasia after intravascular intervention are provided. In particular, the present invention provides luminal prostheses which allow for sustained or controlled release of at least one therapeutic capable agent with increased efficacy to selected locations within a patient's vasculature to reduce restenosis. An intraluminal prosthesis may comprise an expandable structure and a source adjacent the expandable structure for releasing the therapeutic capable agent into a body lumen to reduce smooth muscle cell proliferation.

OS.CITING REF COUNT: 8 THERE ARE 8 CAPLUS RECORDS THAT CITE THIS RECORD (8 CITINGS)

L11 ANSWER 3 OF 21 CAPLUS COPYRIGHT 2009 ACS on STN

ACCESSION NUMBER: 2005:471831 CAPLUS

DOCUMENT NUMBER: 143:1254

TITLE: Combinations and methods for treating neoplasms

INVENTOR(S): Yu, Baofa

PATENT ASSIGNEE(S): USA

SOURCE: U.S. Pat. Appl. Publ., 34 pp., Cont.-in-part of U.S. Ser. No. 765,060.

CODEN: USXXCO

DOCUMENT TYPE: Patent

LANGUAGE: English

FAMILY ACC. NUM. COUNT: 2

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 20050118187	A1	20050602	US 2004-973798	20041025
US 20020044919	A1	20020418	US 2001-765060	20010117 <--
US 6811788	B2	20041102		
PRIORITY APPLN. INFO.:			US 2000-177024P	P 20000119
			US 2001-765060	A2 20010117

AB Methods for treating neoplasms, tumors and cancers, using one or more haptens and coagulation agents or treatments, alone or in combination with other anti-neoplastic agents or treatments, are provided. Also provided are combinations, and kits containing the combinations for effecting the therapy.

OS.CITING REF COUNT: 10 THERE ARE 10 CAPLUS RECORDS THAT CITE THIS RECORD (11 CITINGS)

L11 ANSWER 4 OF 21 CAPLUS COPYRIGHT 2009 ACS on STN

ACCESSION NUMBER: 2002:271945 CAPLUS
DOCUMENT NUMBER: 136:304044
TITLE: Drug complex for treatment of metastatic prostate cancer
INVENTOR(S): D'Amico, Anthony V.; Bubley, Glenn J.; Jebaratnam, David J.; Weinberg, James S.
PATENT ASSIGNEE(S): JCRT Radiation Oncology Support Services, Inc., USA
SOURCE: U.S., 15 pp., Cont.-in-part of U.S. Ser. No. 3,838, abandoned.
CODEN: USXXAM
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 2
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 6368598	B1	20020409	US 1998-110822	19980706 <--
WO 2000001419	A1	20000113	WO 1999-US15126	19990706 <--
W:	AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO			
RW:	GH, GM, KE, LS, MW, SD, SL, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG			
AU 9950897	A	20000124	AU 1999-50897	19990706 <--
US 20030035804	A1	20030220	US 2002-119417	20020409
US 20050233948	A1	20051020	US 2005-102277	20050408
PRIORITY APPLN. INFO.:			US 1996-713114	B1 19960916
			US 1998-3838	B2 19980107
			US 1998-110822	A 19980706
			WO 1999-US15126	W 19990706
			US 2002-119417	A3 20020409

AB A drug complex for delivery of a drug or other agent to a target cell, comprising a targeting carrier mol. which is selectively distributed to a specific cell type or tissue containing the specific cell type; a linker which is acted upon by a mol. which is present at an effective concentration in the environs of the specific cell type; and a drug or an agent to be delivered to the specific cell type. In particular, a drug complex for delivering a cytotoxic drug to prostate cancer cells, comprising a targeting carrier mol. which is selectively delivered to prostate tissue, bone or both; a peptide which is a substrate for prostate specific antigen; and a cytotoxic drug which is toxic to androgen independent prostate cancer cells.

OS.CITING REF COUNT: 3 THERE ARE 3 CAPLUS RECORDS THAT CITE THIS RECORD (3 CITINGS)

REFERENCE COUNT: 24 THERE ARE 24 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L11 ANSWER 5 OF 21 CAPLUS COPYRIGHT 2009 ACS on STN

ACCESSION NUMBER: 2001:935435 CAPLUS
DOCUMENT NUMBER: 136:84677

TITLE: Methods for enhancing antibody-induced cell lysis and treating cancer
 INVENTOR(S): Weiner, George; Hartmann, Gunther
 PATENT ASSIGNEE(S): University of Iowa Research Foundation, USA
 SOURCE: PCT Int. Appl., 312 pp.
 CODEN: PIXXD2
 DOCUMENT TYPE: Patent
 LANGUAGE: English
 FAMILY ACC. NUM. COUNT: 1
 PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2001097843	A2	20011227	WO 2001-US20154	20010622 <--
WO 2001097843	A3	20030123		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, UZ, VN, YU, ZA, ZW				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
CA 2410371	A1	20011227	CA 2001-2410371	20010622 <--
AU 2001070134	A	20020102	AU 2001-70134	20010622 <--
US 20030026801	A1	20030206	US 2001-888326	20010622 <--
US 7534772	B2	20090519		
EP 1296714	A2	20030402	EP 2001-948684	20010622 <--
EP 1296714	B1	20090826		
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR				
JP 2003535907	T	20031202	JP 2002-503327	20010622 <--
AU 2001270134	B2	20060615	AU 2001-270134	20010622 <--
AT 440618	T	20090915	AT 2001-948684	20010622 <--
AU 2006216542	A1	20061012	AU 2006-216542	20060915
AU 2006216542	B2	20090430		
AU 2009203061	A1	20090820	AU 2009-203061	20090728
AU 2009212978	A1	20091001	AU 2009-212978	20090901
PRIORITY APPLN. INFO.:			US 2000-213346P	P 20000622
			AU 2001-270134	A3 20010622
			WO 2001-US20154	W 20010622
			AU 2006-216542	A3 20060915

AB The invention relates to methods and products for treating cancer . In particular the invention relates to combinations of nucleic acids and antibodies for the treatment and prevention of cancer. The invention also relates to diagnostic methods for screening cancer cells.

OS.CITING REF COUNT: 3 THERE ARE 3 CAPLUS RECORDS THAT CITE THIS RECORD (3 CITINGS)
 REFERENCE COUNT: 2 THERE ARE 2 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L11 ANSWER 6 OF 21 CAPLUS COPYRIGHT 2009 ACS on STN
 ACCESSION NUMBER: 2001:545502 CAPLUS
 DOCUMENT NUMBER: 135:117219
 TITLE: Hapten-coagulation agent-antineoplastic agent combinations for treating neoplasms
 INVENTOR(S): Yu, Baofa
 PATENT ASSIGNEE(S): USA
 SOURCE: PCT Int. Appl., 83 pp.
 CODEN: PIXXD2

DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 2
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2001052868	A1	20010726	WO 2001-US1737	20010118 <--
WO 2001052868	A9	20030116		
W:	AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, UZ, VN, YU, ZA, ZW			
RW:	GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG			
CA 2397598	A1	20010726	CA 2001-2397598	20010118 <--
JP 2004505009	T	20040219	JP 2001-552915	20010118 <--
CN 1273146	C	20060906	CN 2001-806830	20010118 <--
AU 2001230977	B2	20061012	AU 2001-230977	20010118 <--
PRIORITY APPLN. INFO.:			US 2000-177024P	P 20000119
			WO 2001-US1737	W 20010118

AB Methods are provided for treating neoplasms, tumors and cancers, using one or more haptens and coagulation agents or treatments, alone or in combination with other anti-neoplastic agents or treatments. Also provided are combinations, and kits containing the combinations for effecting the therapy.

OS.CITING REF COUNT: 1 THERE ARE 1 CAPLUS RECORDS THAT CITE THIS RECORD (1 CITINGS)

REFERENCE COUNT: 8 THERE ARE 8 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L11 ANSWER 7 OF 21 CAPLUS COPYRIGHT 2009 ACS on STN

ACCESSION NUMBER: 2001:12297 CAPLUS

DOCUMENT NUMBER: 134:99574

TITLE: Treating prostate cancer with anti-ErbB2 antibodies

INVENTOR(S): Agus, David B.; Scher, Howard I.; Sliwkowski, Mark X.

PATENT ASSIGNEE(S): Genentech, Inc., USA; Sloan-Kettering Institute for Cancer Research

SOURCE: PCT Int. Appl., 93 pp.

CODEN: PIXXD2

DOCUMENT TYPE: Patent

LANGUAGE: English

FAMILY ACC. NUM. COUNT: 1

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2001000238	A1	20010104	WO 2000-US17423	20000623 <--
W:	AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, UZ, VN, YU, ZA, ZW			
RW:	GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG			
CA 2383493	A1	20010104	CA 2000-2383493	20000623 <--

EP 1189634	A1	20020327	EP 2000-939992	20000623 <--
EP 1189634	B1	20070228		
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, MC, PT, IE, SI, LT, LV, FI, RO, CY, SE				
BR 2000012195	A	20020723	BR 2000-12195	20000623 <--
JP 2003503361	T	20030128	JP 2001-505945	20000623 <--
AU 779209	B2	20050113	AU 2000-54992	20000623 <--
AT 355079	T	20060315	AT 2000-939992	20000623 <--
US 7041292	B1	20060509	US 2000-602800	20000623 <--
ES 2282120	T3	20071016	ES 2000-939992	20000623 <--
CN 100381172	C	20080416	CN 2000-810866	20000623 <--
ZA 2001010088	A	20020826	ZA 2001-10088	20011207
MX 2001013395	A	20030904	MX 2001-13395	20011219
KR 754049	B1	20070831	KR 2001-716538	20011224
US 20060083739	A1	20060420	US 2005-234586	20050923
US 20090087432	A1	20090402	US 2008-247850	20081008
PRIORITY APPLN. INFO.:			US 1999-141315P	P 19990625
			US 2000-602800	A3 20000623
			WO 2000-US17423	W 20000623
			US 2005-234586	B1 20050923

AB The present application discloses treatment of prostate cancer with anti-ErbB2 antibodies. These antibodies are combined with chemotherapeutic agent, cytokine, antiangiogenic agent, EGFR-targeted drug, antiandrogen, anthracycline antibiotic, etc. for treating androgen-(in)dependent prostate cancer.

OS.CITING REF COUNT: 7 THERE ARE 7 CAPLUS RECORDS THAT CITE THIS RECORD (9 CITINGS)

REFERENCE COUNT: 6 THERE ARE 6 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L11 ANSWER 8 OF 21 CAPLUS COPYRIGHT 2009 ACS on STN

ACCESSION NUMBER: 2000:880923 CAPLUS

DOCUMENT NUMBER: 134:37055

TITLE: Methods and compositions using FGF inhibitors and agonists for modulating cell proliferation and cell death

INVENTOR(S): Au, Jessie L. S.; Wientjes, M. Guillaume

PATENT ASSIGNEE(S): USA

SOURCE: PCT Int. Appl., 143 pp.

CODEN: PIXXD2

DOCUMENT TYPE: Patent

LANGUAGE: English

FAMILY ACC. NUM. COUNT: 1

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
-----	----	-----	-----	-----
WO 2000074634	A2	20001214	WO 2000-US40103	20000605 <--
WO 2000074634	A3	20010823		
WO 2000074634	A9	20020926		
W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, SZ, BE, CY, FR, GR, IE, IT, MC, NL, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
CA 2377385	A1	20001214	CA 2000-2377385	20000605 <--
EP 1206234	A2	20020522	EP 2000-943429	20000605 <--

R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT,
IE, SI, LT, LV, FI, RO, MK, CY, AL

JP 2003503313	T	20030128	JP 2001-501171	20000605 <--
US 6599912	B1	20030729	US 2000-587559	20000605 <--
AU 780454	B2	20050324	AU 2000-57903	20000605 <--
IL 146872	A	20061031	IL 2000-146872	20000605 <--
KR 903243	B1	20090617	KR 2001-715591	20011203
US 20040010001	A1	20040115	US 2003-464018	20030618

PRIORITY APPLN. INFO.:

US 1999-137345P	P	19990603
US 1999-165983P	P	19991117
US 1999-172031P	P	19991223
US 2000-187445P	P	20000307
US 2000-587559	A3	20000605
WO 2000-US40103	W	20000605

AB Methods and compns. for modulating the FGF effect on the sensitivity of malignant and normal cells to anticancer agents are provided. In particular, methods and compns. for inhibiting FGF-induced resistance to a broad spectrum of anticancer agents in solid and soft-tissue tumors, metastatic lesions, leukemia and lymphoma are provided. Preferably, the compns. include at least one FGF inhibitor in combination with a cytotoxic agents, e.g., antimicrotubule agents, topoisomerase I inhibitors, topoisomerase II inhibitors, antimetabolites, mitotic inhibitors, alkylating agents, intercalating agents, agents capable of interfering with a signal transduction pathway (e.g., g., a protein kinase C inhibitor, e.g., an anti-hormone, e.g., an antibody against growth factor receptors), an agent that promote apoptosis and/or necrosis, an interferon, an interleukin, a tumor necrosis factor, and radiation. In other embodiments, methods and composition for protecting a cell in a subject, from one or more of killing, inhibition of growth or division or other damage caused, e.g., by a cytotoxic agent, are provided. Preferably, the method includes administering to the subject an effective amount of at least one FGF agonist, thereby treating the cell, e.g., protecting or reducing the damage to the dividing cell from said cytotoxic agent. FGF gene expression-based methods for diagnosis of proliferative disorders are also disclosed.

OS.CITING REF COUNT: 7 THERE ARE 7 CAPLUS RECORDS THAT CITE THIS RECORD (12 CITINGS)

REFERENCE COUNT: 2 THERE ARE 2 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L11 ANSWER 9 OF 21 CAPLUS COPYRIGHT 2009 ACS on STN

ACCESSION NUMBER: 2000:227537 CAPLUS

DOCUMENT NUMBER: 132:262172

TITLE: Use of neoangiogenesis markers for diagnosis and treatment of tumors

INVENTOR(S): Krause, Werner; Muschick, Peter

PATENT ASSIGNEE(S): Schering Aktiengesellschaft, Germany

SOURCE: PCT Int. Appl., 27 pp.

CODEN: PIXXD2

DOCUMENT TYPE: Patent

LANGUAGE: German

FAMILY ACC. NUM. COUNT: 1

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
-----	---	-----	-----	-----
WO 2000018439	A2	20000406	WO 1999-EP7198	19990929 <--
WO 2000018439	A3	20000914		

W: AE, AL, AM, AU, AZ, BA, BB, BG, BR, BY, CA, CN, CR, CU, CZ, DM,
EE, ES, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KG, KP, KR,
KZ, LC, LK, LR, LS, LT, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL,
PT, RO, RU, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ,

VN, YU, ZA, ZW
RW: AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL,
PT, SE

DE 19845798 A1 20000413 DE 1998-19845798 19980929 <--
PRIORITY APPLN. INFO.: DE 1998-19845798 A 19980929

AB Neoangiogenesis markers (i.e. antibodies or receptors for e.g. vascular endothelial growth factor, placenta growth factor, acidic or basic FGF, transforming growth factor α or β , hepatocyte growth factor, insulin-like growth factor I, glycoprotein B61, protein LERK-1, flk-1 receptor, etc.) or partial sequences thereof and antiangiogenic compds. and factors such as paclitaxel, endostatin, fibronectin peptide, and fumagillin are conjugated with active agents such as chemotherapeutic agents, radiosensitizers, photosensitizers, antibodies, oligonucleotides, radioactive metal complexes, etc., which may be bound to carriers, for treatment of tumors. Likewise, neoangiogenesis markers may be conjugated to diagnostic agents such as MRI, radiog., ultrasound, or near-IR contrast agents for tumor diagnosis. Thus, N',N',N''',N''''-tetrakis(tert-butoxycarboxymethyl)-N''-(hydroxycarboxymethyl)diethylenetriamine was converted to its N-hydroxysuccinimide ester, coupled to a Thy-1 antibody, complexed with 186Re, and injected i.v. into rabbits for detection of implanted VX2 tumors by scintigraphy with a gamma camera.

OS.CITING REF COUNT: 9 THERE ARE 9 CAPLUS RECORDS THAT CITE THIS RECORD
(10 CITINGS)

REFERENCE COUNT: 9 THERE ARE 9 CITED REFERENCES AVAILABLE FOR THIS
RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L11 ANSWER 10 OF 21 CAPLUS COPYRIGHT 2009 ACS on STN

ACCESSION NUMBER: 2000:191189 CAPLUS

DOCUMENT NUMBER: 132:227475

TITLE: Treatment of oncologic tumors with an
injectable formulation of a Golgi apparatus disturbing
agent

INVENTOR(S): Singh, Saira Sayed

PATENT ASSIGNEE(S): Oncopharmaceutical, Inc., USA

SOURCE: PCT Int. Appl., 32 pp.

CODEN: PIXXD2

DOCUMENT TYPE: Patent

LANGUAGE: English

FAMILY ACC. NUM. COUNT: 1

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
-----	----	-----	-----	-----
WO 2000015766	A1	20000323	WO 1999-US21312	19990915 <--
W: AU, CA, JP, KR				
RW: AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE				
CA 2344316	A1	20000323	CA 1999-2344316	19990915 <--
AU 9959253	A	20000403	AU 1999-59253	19990915 <--
EP 1114144	A1	20010711	EP 1999-946955	19990915 <--
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI				
US 6287602	B1	20010911	US 1999-397390	19990915 <--
JP 2002525268	T	20020813	JP 2000-570293	19990915 <--
US 20020012703	A1	20020131	US 2001-912115	20010723 <--
US 6497904	B2	20021224		

PRIORITY APPLN. INFO.: US 1998-100479P P 19980916
US 1999-397390 A1 19990915
WO 1999-US21312 W 19990915

AB Novel pharmaceutical formulations for treating a cellular proliferative
disease are provided comprising: a therapeutically effective amount of a

Golgi apparatus disturbing agent; a biocompatible carrier; and a solvent. In preferred formulations, the Golgi apparatus disturbing agent is brefeldin A (BFA) and the biocompatible carrier is a polymer such as chitin or chitosan. Methods of treating cellular proliferative diseases using the pharmaceutical formulations are also described. Nude mice bearing human epithelial (KB-1) tumors were treated with a BFA/chitin/dimethylacetamide composition

OS.CITING REF COUNT: 1 THERE ARE 1 CAPLUS RECORDS THAT CITE THIS RECORD (1 CITINGS)
REFERENCE COUNT: 4 THERE ARE 4 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L11 ANSWER 11 OF 21 CAPLUS COPYRIGHT 2009 ACS on STN

ACCESSION NUMBER: 2000:161302 CAPLUS

DOCUMENT NUMBER: 132:203179

TITLE: Anti-endotoxic, antimicrobial, and cytotoxic cationic peptides and methods of use

INVENTOR(S): Hancock, Robert E. W.; Gough, Monisha A.; Patrzykat, Aleksander; Woods, Donald; Jia, Xiaoyan

PATENT ASSIGNEE(S): University of British Columbia, Can.

SOURCE: PCT Int. Appl., 116 pp.

CODEN: PIXXD2

DOCUMENT TYPE: Patent

LANGUAGE: English

FAMILY ACC. NUM. COUNT: 1

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2000012528	A1	20000309	WO 1999-US19646	19990827 <--
W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU, CZ, DE, DK, DM, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZA, ZW				
RW: GH, GM, KE, LS, MW, SD, SL, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
US 6288212	B1	20010911	US 1998-143124	19980828 <--
CA 2341340	A1	20000309	CA 1999-2341340	19990827 <--
AU 9957890	A	20000321	AU 1999-57890	19990827 <--
AU 758698	B2	20030327		
EP 1107976	A1	20010620	EP 1999-945253	19990827 <--
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO				
US 20030096949	A1	20030522	US 2001-908139	20010717 <--
US 6818407	B2	20041116		
AU 2003204938	A1	20030724	AU 2003-204938	20030625
US 20040186272	A1	20040923	US 2004-823425	20040412
US 20080009445	A1	20080110	US 2007-707813	20070215
PRIORITY APPLN. INFO.:			US 1998-143124	A1 19980828
			AU 1999-57890	A3 19990827
			WO 1999-US19646	W 19990827
			US 2001-908139	A3 20010717
			US 2004-823425	B1 20040412

AB A class of cationic peptides having antimicrobial activity is provided. Exemplary peptides of the invention include KWKSFIKKLTSAKKVVTAKPLALIS and KGWGSFFKKAHVKGKHAALTHYL. Also provided are methods for inhibiting the growth of bacteria utilizing the peptides of the invention. Such methods are useful for the treatment of respiratory infections, e.g. in cystic fibrosis patients. Such methods are further useful for accelerating wound healing. Also disclosed is use of the peptides in

inhibiting the growth of a eukaryotic cell, e.g. a neoplastic cell, and in inhibiting cell proliferation-associated disorders. Transgenic animals, e.g. fish, having a transgene encoding a peptide of the invention are also disclosed.

OS.CITING REF COUNT: 5 THERE ARE 5 CAPLUS RECORDS THAT CITE THIS RECORD (5 CITINGS)
 REFERENCE COUNT: 4 THERE ARE 4 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L11 ANSWER 12 OF 21 CAPLUS COPYRIGHT 2009 ACS on STN

ACCESSION NUMBER: 2000:34771 CAPLUS

DOCUMENT NUMBER: 132:98123

TITLE: Drug complex for targeted treatment of androgen-independent metastatic prostate cancer

INVENTOR(S): D'Amico, Anthony V.; Bubley, Glenn J.; Jebaratnam, David J.; Weinberg, James S.

PATENT ASSIGNEE(S): Ness Medical Center, USA

SOURCE: PCT Int. Appl., 35 pp.

CODEN: PIXXD2

DOCUMENT TYPE: Patent

LANGUAGE: English

FAMILY ACC. NUM. COUNT: 2

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2000001419	A1	20000113	WO 1999-US15126	19990706 <--
W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO				
RW: GH, GM, KE, LS, MW, SD, SL, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
US 6368598	B1	20020409	US 1998-110822	19980706 <--
AU 9950897	A	20000124	AU 1999-50897	19990706 <--
PRIORITY APPLN. INFO.:			US 1998-110822	A 19980706
			US 1996-713114	B1 19960916
			US 1998-3838	B2 19980107
			WO 1999-US15126	W 19990706

AB A drug complex for delivery of a drug or other agent to a target cell, comprising a targeting carrier mol. which is selectively distributed to a specific cell type or tissue containing the specific cell type; a linker which is acted upon by a mol. which is present at an effective concentration in the environs of the specific cell type; and a drug or an agent to be delivered to the specific cell type. In particular, a drug complex for delivering a cytotoxic drug to prostate cancer cells, comprising a targeting carrier mol. which is selectively delivered to prostate tissue, bone or both; a peptide which is a substrate for prostate specific antigen; and a cytotoxic drug which is toxic to androgen independent prostate cancer cells.

OS.CITING REF COUNT: 3 THERE ARE 3 CAPLUS RECORDS THAT CITE THIS RECORD (3 CITINGS)
 REFERENCE COUNT: 6 THERE ARE 6 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L11 ANSWER 13 OF 21 CAPLUS COPYRIGHT 2009 ACS on STN

ACCESSION NUMBER: 1999:783929 CAPLUS

DOCUMENT NUMBER: 132:18780

TITLE: Compositions comprising antimicrotubule agents for treating or preventing inflammatory diseases

INVENTOR(S): Hunter, William L.
PATENT ASSIGNEE(S): Angiotech Pharmaceuticals, Inc., Can.
SOURCE: PCT Int. Appl., 340 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 3
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9962510	A2	19991209	WO 1999-CA464	19990601 <--
W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, GM, HR, HU, ID, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZA, ZW				
RW: GH, GM, KE, LS, MW, SD, SL, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
US 6495579	B1	20021217	US 1998-88546	19980601 <--
AU 2006220416	A1	20061026	AU 2006-220416	20060920
AU 2006220416	B2	20090205		
PRIORITY APPLN. INFO.:			US 1998-88546	A 19980601
			US 1996-32215P	P 19961202
			US 1997-63087P	P 19971024
			US 1997-980549	A2 19971201
			AU 2004-200715	A3 20040220

AB Methods and compns. for treating or preventing inflammatory diseases, e.g. psoriasis or multiple sclerosis, are provided, comprising the step of delivering to the site of inflammation an antimicrotubule agent, or analog or derivative thereof.

OS.CITING REF COUNT: 8 THERE ARE 8 CAPLUS RECORDS THAT CITE THIS RECORD (8 CITINGS)
REFERENCE COUNT: 4 THERE ARE 4 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L11 ANSWER 14 OF 21 CAPLUS COPYRIGHT 2009 ACS on STN
ACCESSION NUMBER: 1999:753254 CAPLUS
DOCUMENT NUMBER: 132:8996
TITLE: Antimicrobial cationic peptide derivatives of bactenecin
INVENTOR(S): Hancock, Robert E. W.; Wu, Manhong
PATENT ASSIGNEE(S): University of British Columbia, Can.
SOURCE: PCT Int. Appl., 52 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9960016	A2	19991125	WO 1999-CA414	19990520 <--
WO 9960016	A3	20000713		
W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, UZ, VN, YU, ZA, ZW				
RW: GH, GM, KE, LS, MW, SD, SL, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG,				

CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG
 US 6172185 B1 20010109 US 1998-82420 19980520 <--
 AU 9938048 A 19991206 AU 1999-38048 19990520 <--
 PRIORITY APPLN. INFO.: US 1998-82420 A 19980520
 WO 1999-CA414 W 19990520

AB A class of cationic peptides having antimicrobial activity is provided. Exemplary peptides of the invention include RLARIVVIRVAR and RLSRIVVIRVCR. Also provided are methods for inhibiting the growth of bacteria using the peptides of the invention. Methods for inhibiting the growth of eukaryotic cells, e.g. tumor cells, with the peptides of the invention are also disclosed.

OS.CITING REF COUNT: 2 THERE ARE 2 CAPLUS RECORDS THAT CITE THIS RECORD (2 CITINGS)

REFERENCE COUNT: 2 THERE ARE 2 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L11 ANSWER 15 OF 21 CAPLUS COPYRIGHT 2009 ACS on STN

ACCESSION NUMBER: 1999:565900 CAPLUS

DOCUMENT NUMBER: 131:194281

TITLE: Conjugated suramin or derivatives thereof with PEG, polyaspartate or polyglutamate for cancer treatment

INVENTOR(S): Webb, Craig P.; Jeffers, Michael E.; Czerwinski, Gregorz; Michejda, Christopher J.; Vande, Woude George F.

PATENT ASSIGNEE(S): The Government of the United States of America, as Represented by the Secretary, Department of Health and Human Services, USA

SOURCE: PCT Int. Appl., 44 pp.
 CODEN: PIXXD2

DOCUMENT TYPE: Patent

LANGUAGE: English

FAMILY ACC. NUM. COUNT: 1

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9943311	A2	19990902	WO 1999-US4336	19990226 <--
WO 9943311	A3	19991014		

W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZW

RW: GH, GM, KE, LS, MW, SD, SL, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

AU 9927954 A 19990915 AU 1999-27954 19990226 <--

PRIORITY APPLN. INFO.: US 1998-75994P P 19980226
 WO 1999-US4336 W 19990226

AB The present invention provides an assay that identifies compds. which inhibit cleavage of HGF/SF by serum proteases such as uPA, and methods in which such compds. are provided to reaction solns., to cultured cells in vitro, or to a mammal in vivo, to inhibit cleavage of HGF/SF (hepatocyte growth factor/scatter factor) and to inhibit chemical and biol. effects resulting from the activation of c-Met receptor by HGF/SF. The invention also provides methods for modifying suramin and suramin-related polysulfonated compds. that inhibit HGF/SF cleavage, by attaching PEG or polyanions such as polyglutamate or polyaspartate to the compds. to reduce cellular uptake of the compds., thereby reducing their cytotoxicity. Also provided are a pharmaceutical composition containing at least one polysulfonated HGF/SF cleavage-inhibiting compound other than

suramin, and a pharmaceutical composition containing at least one HGF/SF cleavage-inhibiting form of suramin or a suramin-related polysulfonated compound that is modified by conjugation to a chemical moiety that reduces uptake of the compound into cells. The present invention further includes methods wherein such pharmaceutical compns. are administered to a mammal with a tumor that is stimulated to grow by HGF/SF, to inhibit the growth or metastasis of the tumor in the mammal.

OS.CITING REF COUNT: 2 THERE ARE 2 CAPLUS RECORDS THAT CITE THIS RECORD (2 CITINGS)
REFERENCE COUNT: 3 THERE ARE 3 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L11 ANSWER 16 OF 21 CAPLUS COPYRIGHT 2009 ACS on STN

ACCESSION NUMBER: 1998:785662 CAPLUS

DOCUMENT NUMBER: 130:33040

TITLE: Methods using 7-(substituted amino)-9-[(substituted glycy)amido]-6-demethyl-6-deoxytetracyclines for inhibiting angiogenesis, proliferation of endothelial or tumor cells, and tumor growth

INVENTOR(S): Backer, Joseph M.; Bohlen, Peter

PATENT ASSIGNEE(S): American Cyanamid Company, USA

SOURCE: U.S., 12 pp.
CODEN: USXXAM

DOCUMENT TYPE: Patent

LANGUAGE: English

FAMILY ACC. NUM. COUNT: 1

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 5843925	A	19981201	US 1994-354694	19941213 <--
US 5856315	A	19990105	US 1998-84484	19980526 <--
PRIORITY APPLN. INFO.:			US 1994-354694	A3 19941213

OTHER SOURCE(S): MARPAT 130:33040

AB A method is provided for inhibiting angiogenesis and proliferation of endothelial cells by administering an inhibitory amount of a 7-(substituted amino)-9-[(substituted glycy)amido]-6-demethyl-6-deoxytetracycline (Markush included). Also provided is a method for inhibiting proliferation of tumor cells and tumor growth by administering an inhibitory amount of a compound of the invention in combination with a chemotherapeutic agent or radiation therapy. Further provided are compns. containing an effective inhibitory amount of a compound of the invention in a pharmaceutically acceptable carrier.

OS.CITING REF COUNT: 2 THERE ARE 2 CAPLUS RECORDS THAT CITE THIS RECORD (2 CITINGS)

REFERENCE COUNT: 24 THERE ARE 24 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L11 ANSWER 17 OF 21 CAPLUS COPYRIGHT 2009 ACS on STN

ACCESSION NUMBER: 1997:394840 CAPLUS

DOCUMENT NUMBER: 127:76021

ORIGINAL REFERENCE NO.: 127:14365a,14368a

TITLE: Compositions and methods using phenylacetic acid derivatives for therapy and prevention of pathologies, including cancer, AIDS and anemia

INVENTOR(S): Samid, Dvorit

PATENT ASSIGNEE(S): United States Dept. of Health and Human Services, USA

SOURCE: U.S., 61 pp., Cont.-in-part of U.S. Ser. No. 779,774.
CODEN: USXXAM

DOCUMENT TYPE: Patent

LANGUAGE: English

FAMILY ACC. NUM. COUNT: 4

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 5635532	A	19970603	US 1993-135661	19931012 <--
US 6037376	A	20000314	US 1991-779744	19911021 <--
EP 1108427	A2	20010620	EP 2000-126980	19921013 <--
EP 1108427	A3	20040107		
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, IE				
EP 1108428	A2	20010620	EP 2000-126981	19921013 <--
EP 1108428	A3	20040107		
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, IE				
ES 2171400	T3	20020916	ES 1992-922550	19921013 <--
EP 1484058	A2	20041208	EP 2004-15994	19921013 <--
EP 1484058	A3	20050427		
EP 1484058	B1	20081231		
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, IE				
EP 1484059	A2	20041208	EP 2004-15995	19921013 <--
EP 1484059	A3	20050420		
EP 1484059	B1	20080903		
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, IE				
AT 406883	T	20080915	AT 2004-15995	19921013 <--
AT 418975	T	20090115	AT 2004-15994	19921013 <--
ES 2312884	T3	20090301	ES 2004-15995	19921013 <--
ZA 9208140	A	19940421	ZA 1992-8140	19921021 <--
CA 2108963	A1	19950422	CA 1993-2108963	19931021 <--
CA 2108963	C	19990316		
US 5605930	A	19970225	US 1994-207521	19940307 <--
IL 111251	A	20040620	IL 1994-111251	19941011 <--
CA 2173976	A1	19950420	CA 1994-2173976	19941012 <--
CA 2173976	C	20080219		
WO 9510271	A2	19950420	WO 1994-US11492	19941012 <--
WO 9510271	A3	19950622		
W: AM, AT, AU, BB, BG, BR, BY, CA, CH, CN, CZ, DE, DK, EE, ES, FI, GB, GE, HU, JP, KE, KG, KP, KR, KZ, LK, LR, LT, LU, LV, MD, MG, MN, MW, NL, NO, NZ, PL, PT, RO, RU, SD, SE, SI, SK, TJ, TT, UA, US, UZ				
RW: KE, MW, SD, SZ, AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG				
AU 702051	B2	19950504	AU 1994-79737	19941012 <--
AU 9479737	A	19950504		
ZA 9407964	A	19960306	ZA 1994-7964	19941012 <--
EP 725635	A1	19960814	EP 1994-930694	19941012 <--
EP 725635	B1	20041229		
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LI, LU, MC, NL, PT, SE				
JP 09506079	T	19970617	JP 1995-511977	19941012 <--
JP 3628694	B2	20050316		
NZ 275673	A	20000929	NZ 1994-275673	19941012 <--
JP 2001253821	A	20010918	JP 2001-69516	19941012 <--
JP 2003119130	A	20030423	JP 2002-302292	19941012 <--
AT 285760	T	20050115	AT 1994-930694	19941012 <--
EP 1523982	A2	20050420	EP 2004-30912	19941012 <--
EP 1523982	A3	20050427		
EP 1523982	B1	20080312		
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT				
ES 2233931	T3	20050616	ES 1994-930694	19941012 <--
AT 388699	T	20080315	AT 2004-30912	19941012 <--
ES 2303624	T3	20080816	ES 2004-30912	19941012 <--
US 5635533	A	19970603	US 1995-470229	19950606 <--
US 5654333	A	19970805	US 1995-465941	19950606 <--

US 5661179	A	19970826	US 1995-469466	19950606 <--
US 5708025	A	19980113	US 1995-465835	19950606 <--
US 5710178	A	19980120	US 1995-469691	19950606 <--
US 5712307	A	19980127	US 1995-465924	19950606 <--
US 5843994	A	19981201	US 1995-478264	19950607 <--
US 5877213	A	19990302	US 1995-484817	19950607 <--
US 5883124	A	19990316	US 1995-484615	19950607 <--
US 5852056	A	19981222	US 1996-633833	19960410 <--
HK 1067551	A1	20081031	HK 2005-100026	20050104
JP 2005139208	A	20050602	JP 2005-54743	20050228
JP 2005139209	A	20050602	JP 2005-54744	20050228
HK 1077204	A1	20090206	HK 2005-109253	20051020
PRIORITY APPLN. INFO.:			US 1991-779744	A2 19911021
			EP 1992-922550	A3 19921013
			US 1993-135661	A2 19931012
			US 1994-207521	A 19940307
			EP 1994-930694	A3 19941012
			JP 1995-511977	A3 19941012
			JP 2001-69516	A3 19941012
			WO 1994-US11492	W 19941012
			EP 2000-126980	A3 20001208
			EP 2000-126981	A3 20001208

OTHER SOURCE(S): MARPAT 127:76021

AB Compns. and methods are disclosed for treating anemia, cancer, AIDS, or severe β -chain hemoglobinopathies by administering a therapeutically effective amount of phenylacetate or pharmaceutically acceptable derivs. thereof or derivs. thereof alone or in combination or in conjunction with other therapeutic agents. Pharmacol.-acceptable salts alone or in combinations and methods of preventing AIDS and malignant conditions, and inducing cell differentiation are also aspects of this invention. Compds. of the invention include $R_0C(R_1)(R_2)[C(R_3)(R_4)]_nC(O)OH$ [R_0 = (substituted) Ph, (substituted) naphthyl, (substituted) phenoxy, where the substitution is 1-4 halo moieties, OH, lower straight-chain or branched alkyl; R_1, R_2 = H, OH, lower alkoxy, halo, lower straight-chain or branched alkyl; R_3, R_4 = H, lower alkoxy, halo, lower straight-chain or branched alkyl; n = 0-2] and pharmaceutically acceptable salts and mixts. thereof.

OS.CITING REF COUNT: 12 THERE ARE 12 CAPLUS RECORDS THAT CITE THIS RECORD (16 CITINGS)

REFERENCE COUNT: 10 THERE ARE 10 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L11 ANSWER 18 OF 21 CAPLUS COPYRIGHT 2009 ACS on STN

ACCESSION NUMBER: 1996:476838 CAPLUS

DOCUMENT NUMBER: 125:105162

ORIGINAL REFERENCE NO.: 125:19439a,19442a

TITLE: Compositions with adenosine derivatives and deaminase inhibitors for the treatment of parasitic and fungal infections and neoplasms

INVENTOR(S): Mccaffrey, Ronald P.; Wigzell, Hans L. R.; Sugar, Alan M.

PATENT ASSIGNEE(S): University Hospital, USA

SOURCE: PCT Int. Appl., 51 pp.

CODEN: PIXXD2

DOCUMENT TYPE: Patent

LANGUAGE: English

FAMILY ACC. NUM. COUNT: 1

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
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WO 9616664	A1	19960606	WO 1995-US15116	19951130 <--

W: AM, AT, AU, BB, BG, BR, BY, CA, CH, CN, CZ, DE, DK, EE, ES, FI,
GB, GE, HU, IS, JP, KE, KG, KP, KR, KZ, LK, LR, LT, LU, LV, MD,
MG, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, TJ,
TM, TT
RW: KE, LS, MW, SD, SZ, UG, AT, BE, CH, DE, DK, ES, FR, GB, GR, IE,
IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR,
NE, SN, TD, TG

US 5663155	A	19970902	US 1994-351068	19941130 <--
US 5679648	A	19971021	US 1994-351067	19941130 <--
CA 2206511	A1	19960606	CA 1995-2206511	19951130 <--
AU 9642411	A	19960619	AU 1996-42411	19951130 <--
EP 794787	A1	19970917	EP 1995-940768	19951130 <--
EP 794787	B1	20030205		
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LI, LU, MC, NL, PT, SE				
JP 10511345	T	19981104	JP 1995-518891	19951130 <--
AT 232104	T	20030215	AT 1995-940768	19951130 <--
PRIORITY APPLN. INFO.:			US 1994-351067	A 19941130
			US 1994-351068	A 19941130
			WO 1995-US15116	W 19951130

OTHER SOURCE(S): MARPAT 125:105162

AB Compns. are provided which comprise an adenosine derivative and a deaminase inhibitor for the prevention and treatment of fungal and fungal-like infections and parasitic infections by eukaryotic organisms. Parasitic infections which are treatable and preventable with these compns. include malaria, trypanosomiasis, leishmania, toxoplasmosis, sarcocystis, pneumocystis, schistosomiasis, blood flukes and elephantitis. Other infections which are treatable and preventable with these compns. are responsible for fungal diseases such as candidiasis, cryptococcosis, blastomycosis, aspergillosis, paracoccidioidomycosis and coccidioidomycosis, and the fungal-like diseases nocardiosis and actinomycosis. The invention also relates to methods for utilizing these compns. in treatment regiments. Treatments may be either in vivo or in vitro. In vivo treatments involve administration of compns. of the invention to mammals suspected or at risk of being infected with a parasitic or fungal organism. In vitro treatments involve incubation of cells, tissues, biol. products derived from living materials or foods with compns. of the invention to inhibit or prevent further infection. Also disclosed is the treatment or prevention of neoplastic disorders with the adenosine derivs. of the invention.

OS.CITING REF COUNT: 6 THERE ARE 6 CAPLUS RECORDS THAT CITE THIS RECORD (6 CITINGS)

L11 ANSWER 19 OF 21 CAPLUS COPYRIGHT 2009 ACS on STN

ACCESSION NUMBER: 1996:464618 CAPLUS

DOCUMENT NUMBER: 125:105098

ORIGINAL REFERENCE NO.: 125:19431a,19434a

TITLE: Combination cancer chemotherapy
with suramin and a vinca-alkaloid or estramustine

INVENTOR(S): Klohs, Wayne Daniel; Kowal, Charles Dale

PATENT ASSIGNEE(S): Warner-Lambert Company, USA

SOURCE: PCT Int. Appl., 18 pp.

CODEN: PIXXD2

DOCUMENT TYPE: Patent

LANGUAGE: English

FAMILY ACC. NUM. COUNT: 1

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
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WO 9619226	A1	19960627	WO 1995-US14008	19951027 <--
W: CA, JP, MX				
RW: AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE				

US 5597830	A	19970128	US 1994-359488	19941220 <--
CA 2206112	A1	19960627	CA 1995-2206112	19951027 <--
EP 794784	A1	19970917	EP 1995-938973	19951027 <--
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LI, LU, MC, NL, PT, SE				
JP 10511347	T	19981104	JP 1995-519779	19951027 <--
US 5767110	A	19980616	US 1996-763601	19961211 <--
PRIORITY APPLN. INFO.:			US 1994-359488	A 19941220
			WO 1995-US14008	W 19951027

AB Suramin in combination with a vinca alkaloid or estramustine is synergistic for treating cancer. Suramin was synergistic with vinblastine against both human prostate and breast cancer cells in vitro. Addnl., suramin and estramustine were synergistic in breast cancer cells and were additive in activity against prostate cancer cells.

OS.CITING REF COUNT: 3 THERE ARE 3 CAPLUS RECORDS THAT CITE THIS RECORD (4 CITINGS)

L11 ANSWER 20 OF 21 CAPLUS COPYRIGHT 2009 ACS on STN

ACCESSION NUMBER: 1991:400778 CAPLUS
DOCUMENT NUMBER: 115:778
ORIGINAL REFERENCE NO.: 115:155a,158a
TITLE: Covalently-linked complexes and methods for enhanced cytotoxicity and imaging
INVENTOR(S): Anderson, David C.; Morgan, A. Charles; Abrams, Paul G.; Nichols, Everett J.; Fritzberg, Alan R.
PATENT ASSIGNEE(S): NeoRx Corp., USA
SOURCE: Eur. Pat. Appl., 23 pp.
CODEN: EPXXDW
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
-----	----	-----	-----	-----
EP 359347	A2	19900321	EP 1989-250014	19890814 <--
EP 359347	A3	19900418		
EP 359347	B1	19921223		
R: AT, BE, CH, DE, ES, FR, GB, GR, IT, LI, LU, NL, SE				
US 5135736	A	19920804	US 1988-232337	19880815 <--
US 5169933	A	19921208	US 1989-390241	19890807 <--
CA 1334513	C	19950221	CA 1989-608198	19890811 <--
JP 02124833	A	19900514	JP 1989-209992	19890814 <--
AT 83669	T	19930115	AT 1989-250014	19890814 <--
PRIORITY APPLN. INFO.:			US 1988-232337	A 19880815
			EP 1989-250014	A 19890814

AB Covalently-linked complexes (CLCs) for targeting a defined population of cells comprise a targeting protein (e.g. antibody, hormone, enzyme, etc.), a cytotoxic agent (e.g. radionuclide, toxin, drug, etc.) an enhancing moiety capable of enhancing CLC-target cell interaction (e.g. a translocating/internalizing moiety, an anchoring peptide, membrane-soluble hydrophobic mol., etc.). The CLCs are used to enhance in vivo cytotoxicity and imaging (no data). Translocating peptide, Cys-Gly-Glu-Ala-Ala-Leu-Ala(Glu-Ala-Leu-Ala)4-Glu-Ala-Leu-Glu-Ala-Leu-Ala-NH₂, is conjugated via succinimidyl 4(N-maleimidemethyl)cyclohexane-1-carboxylate (SMCC) to reduced toxin A chain. The conjugate is reacted with iminothiolane to generate further thiol groups which are then bonded to reduced antibody to prepare translocating peptide-ricin A chain-antibody CLC.

OS.CITING REF COUNT: 51 THERE ARE 51 CAPLUS RECORDS THAT CITE THIS RECORD (60 CITINGS)

L11 ANSWER 21 OF 21 CAPLUS COPYRIGHT 2009 ACS on STN

ACCESSION NUMBER: 1991:35991 CAPLUS
DOCUMENT NUMBER: 114:35991
ORIGINAL REFERENCE NO.: 114:6115a,6118a
TITLE: Isolation of endogenous suramin-induced sulfated
glycosaminoglycans, and their use as
anticancer agents in humans
INVENTOR(S): La Rocca, R. V.; Cooper, M. R.; Stein, C. A.; Myers,
C. E.
PATENT ASSIGNEE(S): National Institutes of Health, USA
SOURCE: U. S. Pat. Appl., 27 pp. Avail. NTIS Order No.
PAT-APPL-7-488 105.
CODEN: XAXXAV
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
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US 488105	A0	19900715	US 1990-488105	19900505 <--
AU 9174781	A	19911010	AU 1991-74781	19900305 <--
WO 9113624	A1	19910919	WO 1991-US1235	19910304 <--

W: AU, CA, JP

RW: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LU, NL, SE

PRIORITY APPLN. INFO.: US 1990-488105 A 19900305
WO 1991-US1235 A 19910304

AB The title sulfated glycosaminoglycans (GAGs) are isolated from
suramin-treated patients and purified. Pharmaceutical compns. containing the
sulfated GAGs are also provided. Thus, when the sulfated GAGs were
isolated from patient urine before and immediately after treatment with
suramin, there was a 4-5-fold increase in GAG excretion that occurred with
suramin treatment. Heparan sulfate, isolated from other urinary GAGs,
demonstrated cytotoxic activity against human carcinoma cell
lines SW-13 (adrenal) and LNCaP-FGC (prostate), with 50% inhibition of
colony formation occurring at uronic acid concns. of 25 and 25-50
µg/mL, resp. Com. bovine kidney heparin sulfate was practically devoid
of activity. An injection formulation contained endogenous
suramin-induced heparan sulfate ammonium salt 150 mg and water for
injection 25 mL.

OS.CITING REF COUNT: 1 THERE ARE 1 CAPLUS RECORDS THAT CITE THIS RECORD
(1 CITINGS)

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(FILE 'HOME' ENTERED AT 14:53:10 ON 21 OCT 2009)

FILE 'REGISTRY' ENTERED AT 14:53:23 ON 21 OCT 2009

L1 13 S SURAMIN

FILE 'CAPLUS' ENTERED AT 14:53:45 ON 21 OCT 2009

L2 2137 S L1

L3 629 S L2 AND (?CANCER? OR ?TUMOR? OR ?TUMOUR? OR ?NEOPLASM?)

L4 205 S L3 AND (CYTOTOXIC? OR CHEMOTHERA?)

L5 66 S L4 AND (?POTENTIAT? OR ?ENHANC? OR ?INCREAS?)

L6 11 S L5 AND (KIT OR COMPOSITION)

FILE 'REGISTRY' ENTERED AT 14:56:51 ON 21 OCT 2009

L7 1 S SURAMIN/CN

FILE 'CAPLUS' ENTERED AT 14:57:15 ON 21 OCT 2009

L8 1782 S L7
L9 605 S L8 AND (?CANCER? OR ?TUMOR? OR ?TUMOUR? OR ?NEOPLASM?)
L10 197 S L9 AND (CYTOTOXIC? OR CHEMOTHERA?)
L11 21 S L10 AND AD<20010924

=> s l11 and kit
44707 KIT
42223 KITS
73613 KIT
(KIT OR KITS)
L12 2 L11 AND KIT

=> d l12 1-2 ibib abs

L12 ANSWER 1 OF 2 CAPLUS COPYRIGHT 2009 ACS on STN
ACCESSION NUMBER: 2005:471831 CAPLUS
DOCUMENT NUMBER: 143:1254
TITLE: Combinations and methods for treating
neoplasms
INVENTOR(S): Yu, Baofa
PATENT ASSIGNEE(S): USA
SOURCE: U.S. Pat. Appl. Publ., 34 pp., Cont.-in-part of U.S.
Ser. No. 765,060.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 2
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 20050118187	A1	20050602	US 2004-973798	20041025
US 20020044919	A1	20020418	US 2001-765060	20010117 <--
US 6811788	B2	20041102		

PRIORITY APPLN. INFO.: US 2000-177024P P 20000119
US 2001-765060 A2 20010117

AB Methods for treating neoplasms, tumors and
cancers, using one or more haptens and coagulation agents or
treatments, alone or in combination with other anti-neoplastic agents or
treatments, are provided. Also provided are combinations, and
kits containing the combinations for effecting the therapy.

OS.CITING REF COUNT: 10 THERE ARE 10 CAPLUS RECORDS THAT CITE THIS
RECORD (11 CITINGS)

L12 ANSWER 2 OF 2 CAPLUS COPYRIGHT 2009 ACS on STN
ACCESSION NUMBER: 2001:545502 CAPLUS
DOCUMENT NUMBER: 135:117219
TITLE: Hapten-coagulation agent-antineoplastic agent
combinations for treating neoplasms
INVENTOR(S): Yu, Baofa
PATENT ASSIGNEE(S): USA
SOURCE: PCT Int. Appl., 83 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 2
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2001052868	A1	20010726	WO 2001-US1737	20010118 <--
WO 2001052868	A9	20030116		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN,
 CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR,
 HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT,
 LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU,
 SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, UZ, VN, YU,
 ZA, ZW

RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY,
 DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF,
 BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

CA 2397598 A1 20010726 CA 2001-2397598 20010118 <--
 JP 2004505009 T 20040219 JP 2001-552915 20010118 <--
 CN 1273146 C 20060906 CN 2001-806830 20010118 <--
 AU 2001230977 B2 20061012 AU 2001-230977 20010118 <--

PRIORITY APPLN. INFO.: US 2000-177024P P 20000119
 WO 2001-US1737 W 20010118

AB Methods are provided for treating neoplasms, tumors
 and cancers, using one or more haptens and coagulation agents or
 treatments, alone or in combination with other anti-neoplastic agents or
 treatments. Also provided are combinations, and kits containing the
 combinations for effecting the therapy.

OS.CITING REF COUNT: 1 THERE ARE 1 CAPLUS RECORDS THAT CITE THIS RECORD
 (1 CITINGS)

REFERENCE COUNT: 8 THERE ARE 8 CITED REFERENCES AVAILABLE FOR THIS
 RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

=> d his

(FILE 'HOME' ENTERED AT 14:53:10 ON 21 OCT 2009)

FILE 'REGISTRY' ENTERED AT 14:53:23 ON 21 OCT 2009
 L1 13 S SURAMIN

FILE 'CAPLUS' ENTERED AT 14:53:45 ON 21 OCT 2009

L2 2137 S L1
 L3 629 S L2 AND (?CANCER? OR ?TUMOR? OR ?TUMOUR? OR ?NEOPLASM?)
 L4 205 S L3 AND (CYTOTOXIC? OR CHEMOTHERA?)
 L5 66 S L4 AND (?POTENTIAT? OR ?ENHANC? OR ?INCREAS?)
 L6 11 S L5 AND (KIT OR COMPOSITION)

FILE 'REGISTRY' ENTERED AT 14:56:51 ON 21 OCT 2009

L7 1 S SURAMIN/CN

FILE 'CAPLUS' ENTERED AT 14:57:15 ON 21 OCT 2009

L8 1782 S L7
 L9 605 S L8 AND (?CANCER? OR ?TUMOR? OR ?TUMOUR? OR ?NEOPLASM?)
 L10 197 S L9 AND (CYTOTOXIC? OR CHEMOTHERA?)
 L11 21 S L10 AND AD<20010924
 L12 2 S L11 AND KIT

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 COST IN U.S. DOLLARS

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FULL ESTIMATED COST

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)

SINCE FILE	TOTAL
ENTRY	SESSION
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L13 9817 L7

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FULL ESTIMATED COST	3.27	166.76
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CA SUBSCRIBER PRICE	0.00	-27.88

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COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	15.08	181.84
DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)	SINCE FILE ENTRY	TOTAL SESSION
CA SUBSCRIBER PRICE	0.00	-27.88

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FILE 'EMBASE' ENTERED AT 15:00:36 ON 21 OCT 2009
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S L14
L15 13200 L14

=> s l13 and l15
L16 9817 L13 AND L15

=> dup rem l16
PROCESSING IS APPROXIMATELY 5% COMPLETE FOR L16

PROCESSING IS APPROXIMATELY 12% COMPLETE FOR L16
PROCESSING IS APPROXIMATELY 20% COMPLETE FOR L16
PROCESSING IS APPROXIMATELY 32% COMPLETE FOR L16
PROCESSING IS APPROXIMATELY 39% COMPLETE FOR L16
PROCESSING IS APPROXIMATELY 46% COMPLETE FOR L16
PROCESSING IS APPROXIMATELY 54% COMPLETE FOR L16
PROCESSING IS APPROXIMATELY 62% COMPLETE FOR L16
PROCESSING IS APPROXIMATELY 69% COMPLETE FOR L16
PROCESSING IS APPROXIMATELY 78% COMPLETE FOR L16
PROCESSING IS APPROXIMATELY 85% COMPLETE FOR L16
PROCESSING IS APPROXIMATELY 94% COMPLETE FOR L16
PROCESSING COMPLETED FOR L16
L17 6551 DUP REM L16 (3266 DUPLICATES REMOVED)

=> s l17 and (?cancer? or ?tumor? or ?tumour? or ?neoplasm?)
L18 1752 L17 AND (?CANCER? OR ?TUMOR? OR ?TUMOUR? OR ?NEOPLASM?)

=> s l18 and pd<20010924
2 FILES SEARCHED...
L19 1247 L18 AND PD<20010924

=> s l19 and (?potentiat? or ?enhanc? or ?increas?)
L20 298 L19 AND (?POTENTIAT? OR ?ENHANC? OR ?INCREAS?)

=> s l20 and (carboplatin or paclitaxel or docetaxel or gemcitabine or
5-fluorouracil)
L21 32 L20 AND (CARBOPLATIN OR PACLITAXEL OR DOCETAXEL OR GEMCITABINE
OR 5-FLUOROURACIL)

=> s l21 and (kit or composition)
L22 0 L21 AND (KIT OR COMPOSITION)

=> d l21 1-32 ibib abs

L21 ANSWER 1 OF 32 MEDLINE on STN
ACCESSION NUMBER: 2001514410 MEDLINE
DOCUMENT NUMBER: PubMed ID: 11561988
TITLE: Treatment of hormone refractory prostate cancer.
AUTHOR: Knox J J; Moore M J
CORPORATE SOURCE: Department of Medical Oncology and Hematology, Princess
Margaret Hospital, University Health Network, University of
Toronto, Canada.
SOURCE: Seminars in urologic oncology, (2001 Aug) Vol.
19, No. 3, pp. 202-11. Ref: 58
Journal code: 9514993. ISSN: 1081-0943.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200201
ENTRY DATE: Entered STN: 20 Sep 2001
Last Updated on STN: 25 Jan 2002
Entered Medline: 3 Jan 2002

AB Hormone refractory prostate cancer (HRPC) is a difficult
clinical problem. These patients are intolerant of aggressive cytotoxic
therapies because of their age and poor performance status. Systemic
chemotherapy, whether administered as single agents or in multidrug
combinations, has not been shown to prolong survival. It is only recently
that palliative endpoints, such as quality of life analyses, have been
formally evaluated in the clinical trials of HRPC. As a result,
mitoxantrone plus prednisone has been demonstrated to be a useful

palliative therapy that provides improvements in pain and quality of life for approximately 40% of those treated. Other promising regimens, such as the estramustine combinations or docetaxel, are currently undergoing phase III trials designed to prove superiority to mitoxantrone plus prednisone. Suramin has been extensively studied, but due to its poor activity seen in recent randomized trials, as well as the toxicity and inconvenience, it will likely not be further developed in HRPC. In recent years, there has been a tremendous increase in the development of biological targets for cancer therapy and a number of these are in early trials for HRPC. Given the relative insensitivity of prostate cancer to cytotoxic agents, this area holds much potential.

L21 ANSWER 2 OF 32 MEDLINE on STN
 ACCESSION NUMBER: 2001462179 MEDLINE
 DOCUMENT NUMBER: PubMed ID: 11507065
 TITLE: Nontoxic doses of suramin enhance activity of paclitaxel against lung metastases.
 AUTHOR: Song S; Wientjes M G; Walsh C; Au J L
 CORPORATE SOURCE: College of Pharmacy and James Cancer Hospital and Solove Research Institute, Ohio State University, Columbus, Ohio 43210, USA.
 CONTRACT NUMBER: R01CA78577 (United States NCI NIH HHS)
 R37CA49816 (United States NCI NIH HHS)
 SOURCE: Cancer research, (2001 Aug 15) Vol. 61, No. 16, pp. 6145-50.
 Journal code: 2984705R. ISSN: 0008-5472.
 PUB. COUNTRY: United States
 DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
 (RESEARCH SUPPORT, U.S. GOV'T, P.H.S.)
 LANGUAGE: English
 FILE SEGMENT: Priority Journals
 ENTRY MONTH: 200109
 ENTRY DATE: Entered STN: 20 Aug 2001
 Last Updated on STN: 10 Sep 2001
 Entered Medline: 6 Sep 2001

AB We recently reported that acidic (aFGF) and basic (bFGF) fibroblast growth factors confer a broad spectrum chemoresistance in solid tumors, and that suramin, an inhibitor of multiple growth factors including aFGF and bFGF, enhanced the in vitro antitumor activity of several anticancer drugs including paclitaxel (Song, S., et al., Proc. Natl. Acad. Sci. USA, 97: 8658-8663, 2000). The present study investigated in vitro and in vivo interaction between paclitaxel and suramin, using human PC3-LN cells which, upon i.v. injection into immunodeficient mice, yielded lung metastases in 100% of animals. In in vitro studies, conditioned medium (CM) obtained from histocultures of rat lung metastases induced a 3-fold resistance. The addition of suramin had no effect in the absence of CM but reversed the CM-induced resistance; calculations based on the IC(50) values indicate a complete reversal in the presence of <20 microM suramin. Analysis by the combination index method indicates a synergistic interaction between paclitaxel and suramin. In in vivo studies, animals with well-established lung metastases (at least five nodules of 1 mm in diameter) were treated i.v. with paclitaxel (15 mg/kg) and suramin (10 mg/kg) administered twice weekly for 3 weeks. Single-drug therapy with paclitaxel or suramin did not reduce body weight. Suramin alone had no antitumor activity. Paclitaxel alone reduced the tumor size by approximately 75%, reduced the density of nonapoptotic cells by approximately 70% in residual tumors, and enhanced the fraction of apoptotic cells by approximately 3-fold. The addition of

suramin to paclitaxel therapy enhanced the antitumor effect, resulting in an additional 5-fold reduction of tumor size, an additional 9-fold reduction of the density of nonapoptotic cells, and an additional 30% increase in the apoptotic cell fraction. These data indicate significant enhancement of the efficacy of paclitaxel by suramin and support the use of nontoxic doses of suramin with paclitaxel in the treatment of lung cancer.

L21 ANSWER 3 OF 32 MEDLINE on STN
ACCESSION NUMBER: 2001090757 MEDLINE
DOCUMENT NUMBER: PubMed ID: 11143501
TITLE: [Peripheral nervous system neurotoxicity secondary to chemotherapy treatment]].
Neurotoxicidad en el sistema nervioso periferico secundaria a tratamiento con quimioterapia.
AUTHOR: Iniguez C; Larrode P; Mayordomo J I; Mauri J A; Tres A; Morales F
CORPORATE SOURCE: Servicio de Neurologia, Hospital Clinico Universitario, Juan Bosco, 15, 50009 Zaragoza.. ciniguezmn@nacom.es
SOURCE: Neurologia (Barcelona, Spain), (2000 Oct) Vol. 15, No. 8, pp. 343-51. Ref: 56
Journal code: 9005460. ISSN: 0213-4853.
PUB. COUNTRY: Spain
DOCUMENT TYPE: (ENGLISH ABSTRACT)
Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
LANGUAGE: Spanish
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200101
ENTRY DATE: Entered STN: 22 Mar 2001
Last Updated on STN: 22 Mar 2001
Entered Medline: 25 Jan 2001

AB Peripheral neurotoxicity is a crucial side effect of chemotherapeutic agents. It is the only situation where there is no preventive treatment. Neuromuscular toxicity has become the major dose limiting side effect for many chemotherapeutic agents. The iatrogenic toxic neuropathy is a growing neurologic problem, as cancer patients are being treated with increasing doses of chemotherapy drugs. Major advances in cancer treatment have resulted from the use of drug combinations; for some combinations this raises the possibility of synergistic neurotoxicity. The following report reviews the SNP toxicities encountered with cisplatin, vincristine, taxanes and others, and methods to minimize the deleterious effect of chemotherapeutic agents.

L21 ANSWER 4 OF 32 MEDLINE on STN
ACCESSION NUMBER: 2000406964 MEDLINE
DOCUMENT NUMBER: PubMed ID: 10890892
TITLE: Fibroblast growth factors: an epigenetic mechanism of broad spectrum resistance to anticancer drugs.
AUTHOR: Song S; Wientjes M G; Gan Y; Au J L
CORPORATE SOURCE: College of Pharmacy and James Cancer Hospital and Solove Research Institute, Ohio State University, Columbus, OH 43210, USA.
CONTRACT NUMBER: R01CA63363 (United States NCI NIH HHS)
R01CA78577 (United States NCI NIH HHS)
R37CA49816 (United States NCI NIH HHS)
SOURCE: Proceedings of the National Academy of Sciences of the United States of America, (2000 Jul 18) Vol. 97, No. 15, pp. 8658-63.
Journal code: 7505876. ISSN: 0027-8424.
Report No.: NLM-PMC27004.

PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, U.S. GOV'T, P.H.S.)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200008
ENTRY DATE: Entered STN: 1 Sep 2000
Last Updated on STN: 1 Sep 2000
Entered Medline: 24 Aug 2000

AB Based on the observation that removal of tumors from metastatic organs reversed their chemoresistance, we hypothesized that chemoresistance is induced by extracellular factors in tumor-bearing organs. By comparing chemosensitivity and proteins in different tumors (primary vs. metastases) and different culture systems (tumor fragment histocultures vs. monolayer cultures derived from the same tumor), we found elevated levels of acidic (aFGF) and basic (bFGF) fibroblast growth factors in the conditioned medium (CM) of solid and metastatic tumors. These CM induced broad spectrum resistance to drugs with diverse structures and action mechanisms (paclitaxel, doxorubicin, 5-fluorouracil). Inhibition of bFGF by mAb and its removal by immunoprecipitation resulted in complete reversal of the CM-induced chemoresistance, whereas inhibition/removal of aFGF resulted in partial reversal. Using CM that had been depleted of aFGF and/or bFGF and subsequently reconstituted with respective human recombinant proteins, we found that bFGF but not aFGF induced chemoresistance whereas aFGF amplified the bFGF effect. aFGF and bFGF fully accounted for the CM effect, indicating these proteins as the underlying mechanism of the chemoresistance. The FGF-induced resistance was not due to reduced intracellular drug accumulation or altered cell proliferation. We further showed that an inhibitor of aFGF/bFGF (suramin) enhanced the in vitro and in vivo activity of chemotherapy, resulting in shrinkage and eradication of well established human lung metastases in mice without enhancing toxicity. These results indicate elevated levels of extracellular aFGF/bFGF as an epigenetic mechanism by which cancer cells elude cytotoxic insult by chemotherapy, and provide a basis for designing new treatment strategies.

L21 ANSWER 5 OF 32 MEDLINE on STN
ACCESSION NUMBER: 1998369155 MEDLINE
DOCUMENT NUMBER: PubMed ID: 9701726
TITLE: Apoptotic effects of different drugs on cultured retinoblastoma Y79 cells.
AUTHOR: Lauricella M; Giuliano M; Emanuele S; Vento R; Tesoriere G
CORPORATE SOURCE: Institute of Biological Chemistry, University of Palermo, Policlinico, Palermo, Italy.
SOURCE: Tumour biology : the journal of the International Society for Oncodevelopmental Biology and Medicine, (1998)
Vol. 19, No. 5, pp. 356-63.
Journal code: 8409922. ISSN: 1010-4283.
PUB. COUNTRY: Switzerland
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199809
ENTRY DATE: Entered STN: 6 Oct 1998
Last Updated on STN: 29 Jan 1999
Entered Medline: 18 Sep 1998

AB This paper deals with the apoptotic effect exerted in human retinoblastoma Y79 cells by a number of compounds. A remarkable effect was observed after treatment with DNA-damaging agents, such as camptothecin, etoposide,

cisplatin and carboplatin; camptothecin was found to be the most efficacious. Treatment with these compounds induced the appearance of morphological features of apoptosis in the cells together with the distinct fragmentation of DNA, as shown by agarose gel electrophoresis. These effects were also accompanied by a remarkable increase in the level of p53. Many other compounds, which are not DNA-damaging agents, induced the morphological features of apoptosis but none of them were capable of increasing the level of p53. Among these compounds, Taxol, suramin and sodium butyrate also stimulated the oligonucleosomal fragmentation of DNA, while C2-ceramide, a cell-permeable analogue of ceramide, and vitamin D3 were not effective in the induction of DNA laddering in Y79 cells. Apoptosis was dependent on macromolecular synthesis with all the compounds tested.

L21 ANSWER 6 OF 32 MEDLINE on STN
ACCESSION NUMBER: 1997338728 MEDLINE
DOCUMENT NUMBER: PubMed ID: 9195288
TITLE: Preclinical studies of the combination of angiogenic inhibitors with cytotoxic agents.
AUTHOR: Kakeji Y; Teicher B A
CORPORATE SOURCE: Dana-Farber Cancer Institute, Boston, MA 021150, USA.
SOURCE: Investigational new drugs, (1997) Vol. 15, No. 1, pp. 39-48.
Journal code: 8309330. ISSN: 0167-6997.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199708
ENTRY DATE: Entered STN: 8 Sep 1997
Last Updated on STN: 6 Feb 1998
Entered Medline: 26 Aug 1997

AB TNP-740, minocycline, suramin and genistein have demonstrated antiangiogenic activity in various experimental systems. The effect of these agents alone and in two agent combinations on the number of intratumoral vessels and response to cytotoxic anticancer therapies was assessed in animals bearing the Lewis lung carcinoma. Treatment with each of the antiangiogenic agents alone and in two agent combinations decreased the number of intratumoral vessels visualized by CD31 or Factor VIII staining to 30% to 50% of the number in the untreated control tumors. In general, the antiangiogenic agents are more effective adjuvants to cytotoxic therapies when used as two agent combinations than as single agents. The most effective antiangiogenic combinations were: TNP-470/minocycline > TNP-470/genistein > TNP-470/suramin. The increases in the response of the primary tumor to cyclophosphamide, adriamycin, CDDP, BCNU, x-rays or 5-fluorouracil and the lung metastases occur to about the same level with the addition of antiangiogenic agents to the therapies. With the treatment combination TNP-470/minocycline/cyclophosphamide 40% of the animals were cured. The results of these studies indicate that antiangiogenic agents can be very useful additions to treatment regimens for solid tumors.

L21 ANSWER 7 OF 32 MEDLINE on STN
ACCESSION NUMBER: 1995265050 MEDLINE
DOCUMENT NUMBER: PubMed ID: 7746278
TITLE: Altered topoisomerase I and II activities in suramin-resistant lung fibrosarcoma cells.
AUTHOR: Lelievre S; Benchokroun Y; Larsen A K
CORPORATE SOURCE: Department of Structural Biology and Pharmacology, CNRS URA 147, Institute Gustave Roussy, Villejuif, France.
SOURCE: Molecular pharmacology, (1995 May) Vol. 47, No.

5, pp. 898-906.
Journal code: 0035623. ISSN: 0026-895X.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199506
ENTRY DATE: Entered STN: 21 Jun 1995
Last Updated on STN: 3 Feb 1997
Entered Medline: 12 Jun 1995

AB To better understand the molecular basis for the cytotoxic effects of suramin, we have developed suramin-resistant DC-3F/SU 1000 cells by continuous exposure of fibrosarcoma cells to increasing concentrations of suramin. The suramin resistance (approximately 10-fold) is not associated with changes in uptake or intracellular distribution of the drug. The sensitivity to actinomycin D, cytarabine, aphidicolin, hydroxyurea, vincristine, and 5-fluorouracil is unaltered. In contrast, DC-3F/SU 1000 cells are about 2-fold resistant to classical DNA topoisomerase II inhibitors such as doxorubicin, amsacrine, and etoposide, whereas the cells are 1.5-fold more sensitive to the topoisomerase I inhibitor camptothecin. The cross-resistance to topoisomerase II inhibitors occurred earlier than the collateral sensitivity to camptothecin. Amsacrine- and etoposide-induced DNA-protein complex formation is reduced about 2-fold in DC-3F/SU 1000 cells, compared with DC-3F cells, whereas camptothecin-induced DNA-protein complex formation is increased 1.5-fold. Western blot analysis of cellular lysates from the two cell lines shows no significant differences in the level of topoisomerase II, whereas the level of topoisomerase I is increased 2.5-fold in DC-3F/SU 1000 cells. The catalytic activities of topoisomerases I and II in nuclear extracts from DC-3F/SU 1000 cells are both about 2-fold higher than those in extracts from DC-3F cells, whereas amsacrine- and etoposide-induced DNA-protein complex formation is comparable between the two cell lines. Taken together, our results support the involvement of DNA topoisomerases in the cytotoxic activity of suramin. We further believe that the DC-3F/SU 1000 cells may be a useful model for the elucidation of factors that lead to low, clinically relevant, levels of resistance to topoisomerase II inhibitors.

L21 ANSWER 8 OF 32 MEDLINE on STN
ACCESSION NUMBER: 1995134507 MEDLINE
DOCUMENT NUMBER: PubMed ID: 7833116
TITLE: The synergistic and antagonistic effects of cytotoxic and biological agents on the in vitro antitumour effects of suramin.
AUTHOR: Lopez Lopez R; van Rijswijk R E; Wagstaff J; Pinedo H M; Peters G J
CORPORATE SOURCE: Department of Oncology, Free University Hospital, Amsterdam, The Netherlands.
SOURCE: European journal of cancer (Oxford, England : 1990), (1994) Vol. 30A, No. 10, pp. 1545-9.
Journal code: 9005373. ISSN: 0959-8049.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199502
ENTRY DATE: Entered STN: 14 Mar 1995
Last Updated on STN: 3 Feb 1997
Entered Medline: 28 Feb 1995

AB Suramin has shown antitumour activity in vitro and in vivo. At plasma levels higher than 200 microM there is, however, excessive toxicity. We have, therefore, attempted to improve the antitumour effects of suramin in vitro by combining it with several other antitumour agents. The MCF-7 mammary carcinoma and PC3 prostate cancer cell lines were exposed continuously to suramin and the other agents for 6 days. The sulphorhodamine B (SRB) assay was used for the assessment of growth inhibition. The dose-response interactions were evaluated using the median-effect analysis with the Chou and Talalay computer programme. In the MCF-7 cell line, the combination of suramin plus doxorubicin (DXR), cisplatin (CDDP), 5-fluorouracil (5-FU) or tumour necrosis factor (TNF) resulted in synergistic growth inhibition, whilst its combination with miltefosine (HPC) was antagonistic. In the PC-3 cell line, suramin plus CDDP or TNF was synergistic, whilst its combination with DXR, 5-FU and HPC was antagonistic. All tested combinations with interferon-alpha (IFN-alpha), interferon-gamma (IFN-gamma) and with the combination of both IFN-alpha+IFN-gamma were not synergistic. The synergistic effect of suramin with DXR was schedule dependent. Pretreatment (addition of DXR on day 1 and suramin on days 2-5) was additive at the IC50 level, in both cell lines. Addition of DXR at day 5 was more effective than simultaneous exposure. We found a synergistic effect for the combination of suramin with CDDP and TNF in both cell lines. In addition the combination with DXR and 5-FU was synergistic in MCF-7. Sequential administration of DXR-suramin or suramin-DXR increased the growth inhibition.

L21 ANSWER 9 OF 32 MEDLINE on STN
ACCESSION NUMBER: 1994220841 MEDLINE
DOCUMENT NUMBER: PubMed ID: 7513218
TITLE: Promising new developments in the systemic treatment of ovarian cancer.
AUTHOR: Reed E
CORPORATE SOURCE: Medical Ovarian Cancer Section, Medicine Branch, National Cancer Institute, Bethesda, Maryland 20892.
SOURCE: Journal of the Association for Academic Minority Physicians : the official publication of the Association for Academic Minority Physicians, (1994) Vol. 5, No. 1, pp. 16-21. Ref: 30
Journal code: 9113765. ISSN: 1048-9886.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199405
ENTRY DATE: Entered STN: 13 Jun 1994
Last Updated on STN: 29 Jan 1996
Entered Medline: 27 May 1994

AB Advanced-stage cancer of the ovary is the most lethal of gynecologic malignancies, affecting African-American and white women with approximately equal frequency. In large part, ovarian cancer's lethality is due to the fact that most women are diagnosed with disease that is widespread throughout the abdomen and pelvis. This article reviews recent developments in the identification of new treatment approaches to ovarian cancer. Discussion focuses on current drug development activities of the Medical Ovarian Cancer Section of the National Cancer Institute, with reference to pertinent literature from other institutions. The drugs discussed are in clinical trials as of this writing. They include paclitaxel, an agent with a novel molecular mechanism of action; colony-stimulating

factors, which enhance the therapeutic index of cytotoxic agents; and the antiproliferative agents suramin and carboxyamidotriazole.

L21 ANSWER 10 OF 32 EMBASE COPYRIGHT (c) 2009 Elsevier B.V. All rights reserved on STN

ACCESSION NUMBER: 2002093433 EMBASE
TITLE: DNA topoisomerases as targets for anticancer drugs.
AUTHOR: Topcu, Z. (correspondence)
CORPORATE SOURCE: Dept. of Pharmaceut. Biotechnology, Faculty of Pharmacy, Ege University, 35100 Izmir, Turkey. ztopcu@bornova.ege.edu.tr
SOURCE: Journal of Clinical Pharmacy and Therapeutics, (2001) Vol. 26, No. 6, pp. 405-416.
Refs: 90
ISSN: 0269-4727 CODEN: JCPTED
COUNTRY: United Kingdom
DOCUMENT TYPE: Journal; General Review; (Review)
FILE SEGMENT: 016 Cancer
030 Clinical and Experimental Pharmacology
037 Drug Literature Index
038 Adverse Reactions Titles
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 21 Mar 2002
Last Updated on STN: 21 Mar 2002

AB DNA topoisomerases are essential enzymes that regulate the conformational changes in DNA topology by catalysing the concerted breakage and rejoining of DNA strands during normal cellular growth. Over the past few years there has been considerable pharmacological interest in these enzymes because inhibitors of DNA topoisomerases represent a major class of anticancer drugs. This review highlights topoisomerase-targeting drugs that have shown promising anticancer activities. The mechanisms by which those drugs interfere with the catalytic cycles of type I and type II DNA topoisomerases and the factors involved in the development of resistance to these drugs are discussed.

L21 ANSWER 11 OF 32 EMBASE COPYRIGHT (c) 2009 Elsevier B.V. All rights reserved on STN

ACCESSION NUMBER: 2002033367 EMBASE
TITLE: The treatment challenge of hormone-refractory prostate cancer.
AUTHOR: Kish, Julie A., Dr. (correspondence); Bukkapatnam, Raviender; Palazzo, Felipe
CORPORATE SOURCE: H. L. Moffitt Cancer Ctr./Res. Inst., MCC-H and NPROG, 12902 Magnolia Drive, Tampa, FL 33612, United States. KishJA@moffitt.usf.edu
SOURCE: Cancer Control, (2001) Vol. 8, No. 6, pp. 487-495.
Refs: 62
ISSN: 1073-2748 CODEN: CACOFD
COUNTRY: United States
DOCUMENT TYPE: Journal; General Review; (Review)
FILE SEGMENT: 016 Cancer
037 Drug Literature Index
038 Adverse Reactions Titles
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 31 Jan 2002
Last Updated on STN: 31 Jan 2002

AB Background: Both the demographics and treatment of hormone-refractory

prostate cancer (HRPC) are changing. Patients are younger and healthier, with fewer comorbidities. The "no treatment until symptoms" approach is disappearing. Chemotherapy is increasingly being utilized. Methods: The authors review the steps involved in hormone management before chemotherapy is considered. The roles for chemotherapy in current clinical trials are examined. Results: Effective hormonal management of the prostate cancer patient incorporates an understanding of the stages of hormone sensitivity and prescribing additional interventions beyond simple castration. Once hormone refractoriness is established, the combination of mitoxantrone and prednisone has become a standard chemotherapeutic approach. New agents such as docetaxel are being tested in phase III trials against mitoxantrone plus prednisone. Conclusions: HRPC is now regarded as a chemotherapy-sensitive tumor. The goals of chemotherapy in HRPC are to decrease PSA level and improve quality of life. New agents and combinations are needed to improve survival.

L21 ANSWER 12 OF 32 EMBASE COPYRIGHT (c) 2009 Elsevier B.V. All rights reserved on STN

ACCESSION NUMBER: 2001363191 EMBASE
 TITLE: [Perspectives of anti-cancer therapy].
 Perspektivni moznosti v protinadorove chemoterapii.
 AUTHOR: Klener, P., Dr. (correspondence)
 CORPORATE SOURCE: I Interni Klinika, I Lekarska Fakulta, UK a VFN, U
 Nemecnice 2, 128 08 Praha 2, Czech Republic. pavel.klener@r
 uk.cuni.cz
 SOURCE: Casopis Lekaru Ceskych, (2001) Vol. 140, No. 19,
 pp. 605-610.
 Refs: 22
 ISSN: 0008-7335 CODEN: CLCEAL
 COUNTRY: Czech Republic
 DOCUMENT TYPE: Journal; General Review; (Review)
 FILE SEGMENT: 016 Cancer
 037 Drug Literature Index
 LANGUAGE: Czech
 SUMMARY LANGUAGE: English; Czech
 ENTRY DATE: Entered STN: 2 Nov 2001
 Last Updated on STN: 2 Nov 2001

AB The unsatisfactory results of current anti-cancer therapies require the search for new drugs and new approaches. The review summarizes different possibilities of future treatments such as antiangiogenesis, inhibition of metastatic cascade, induction of differentiation. The most promising is to the influence signal transduction and to the control cell cycle progression. Increased understanding in the mechanisms driving cellular proliferation emerges novel therapeutics that are more specific and less toxic than classical chemotherapy.

L21 ANSWER 13 OF 32 EMBASE COPYRIGHT (c) 2009 Elsevier B.V. All rights reserved on STN

ACCESSION NUMBER: 2001255890 EMBASE
 TITLE: A reexamination of PSC 833 (Valspodar) as a cytotoxic agent and in combination with anticancer agents.
 AUTHOR: Kreis, W. (correspondence); Budman, D.R.; Calabro, A.
 CORPORATE SOURCE: Don Monti Division of Oncology, North Shore University Hospital, 300 Community Drive, Manhasset, NY 11030, United States.
 SOURCE: Cancer Chemotherapy and Pharmacology, (2001) Vol. 47, No. 1, pp. 78-82.
 Refs: 29
 ISSN: 0344-5704 CODEN: CCPHDZ
 COUNTRY: Germany

DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 016 Cancer
037 Drug Literature Index
005 General Pathology and Pathological Anatomy
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 15 Aug 2001
Last Updated on STN: 15 Aug 2001

AB Background: The cyclosporins have been thought as being mainly immunosuppressive agents which interfere with the function of the MDR pump and thus play a role in resistance to drug anticancer effects. We reexamined their cytotoxicity in defined cell lines both as single agents and in combination with agents which may be of value in human malignant disease. Methods: Cells were grown to confluence following inoculation at 5000-8000 cells/well in 96-well dishes, and growth, patterns and death were determined by an MTT assay. Median effect analysis was used to look for synergy, additive effects, or antagonism between the cyclosporins and drugs with antitumor effects in humans. Results: Cyclosporin A and PSC 833 were found to have cytotoxic activity at clinically achievable concentrations in breast, leukemia, and prostate cell lines. Synergistic or additive effects were demonstrated in all three prostate cell lines when PSC 833 was combined with estramustine, etoposide, ketoconazole, suramin, or vinorelbine in the prostate cancer cell lines. Cell line-selective additive effects or synergism were also identified with bicalutamide, carboplatin, cisplatin, cis-retinoic acid, dexamethasone, 5-fluorouracil, liarozole, and trans-retinoic acid. Conclusions: PSC 833 or cyclosporin alone or in combination with other agents may have an anticancer effect independently of their modulatory action on MDR. Several of the synergistic combinations which are not mediated by the MDR pump need to be tested in vivo for efficacy.

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ACCESSION NUMBER: 2001151899 EMBASE
TITLE: Incidence rate and management of prostate carcinoma.
AUTHOR: Sandblom, G. (correspondence); Varenhorst, E.
CORPORATE SOURCE: Department of Urology, Faculty of Health Sciences,
University Hospital, 581 85 Linköping, Sweden. gabsa@ibk.li
u.se
SOURCE: Biomedicine and Pharmacotherapy, (2001) Vol. 55,
No. 3, pp. 135-143.
Refs: 78

ISSN: 0753-3322 CODEN: BIPHEX

PUBLISHER IDENT.: S 0753-3322(01)00038-5

COUNTRY: France

DOCUMENT TYPE: Journal; Article

FILE SEGMENT: 016 Cancer
028 Urology and Nephrology
030 Clinical and Experimental Pharmacology
037 Drug Literature Index
038 Adverse Reactions Titles

LANGUAGE: English

SUMMARY LANGUAGE: English

ENTRY DATE: Entered STN: 10 May 2001

Last Updated on STN: 10 May 2001

AB The age-standardised incidence of prostate cancer varies more than one hundredfold between the areas with the highest and lowest incidences in the world. In certain areas, in particular the Western countries, the incidence has increased rapidly over the last 20 years. There are several environmental and genetic factors which partly explain these variations, although the incidence probably depends most of

all on the extent to which small latent tumours are detected. As the clinical significance of small tumours is uncertain, the value of early diagnosis and early aggressive treatment is controversial. Randomised trials addressing this question have been initiated and will hopefully provide more evidence-based data in a decade from now. Small localised tumours are managed by radical surgery or radiation therapy. In elderly men or men unfit for operation or radiation therapy surveillance is often preferred. For advanced or metastatic prostate cancers androgen deprivation has been the mainstay of treatment since the early 1940s. Recently, several new treatment strategies have evolved but have not yet been introduced into clinical routine. .COPYRG.T. 2001 Editions scientifiques et medicales Elsevier SAS.

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ACCESSION NUMBER: 2001109350 EMBASE
TITLE: Targeting ceramide metabolism - A strategy for overcoming drug resistance.
AUTHOR: Senchenkov, A.; Litvak, D.A.; Cabot, M.C., Dr. (correspondence)
CORPORATE SOURCE: Breast Cancer Res. Prog./Chemother., John Wayne Cancer Institute, Saint John's Health Center, 2200 Santa Monica Blvd., Santa Monica, CA 90404, United States. cabot@jwci.org
SOURCE: Journal of the National Cancer Institute, (7 Mar 2001) Vol. 93, No. 5, pp. 347-357.
Refs: 150
ISSN: 0027-8874 CODEN: JNCIAM
COUNTRY: United Kingdom
DOCUMENT TYPE: Journal; General Review; (Review)
FILE SEGMENT: 016 Cancer
030 Clinical and Experimental Pharmacology
037 Drug Literature Index
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 12 Apr 2001
Last Updated on STN: 12 Apr 2001

AB Inherent or acquired drug resistance, which frequently characterizes cancer cells, is caused by multiple mechanisms, including dysfunctional metabolism of the lipid second messenger ceramide. Ceramide, the basic structural unit of the sphingolipids, plays a role in activating cell death signals initiated by cytokines, chemotherapeutic agents, and ionizing radiation. Recent discoveries about the metabolism of ceramide suggest that this agent may have an important influence on the effectiveness of various cancer therapeutics. In particular, the cytotoxic effect of chemotherapy is decreased when generation of ceramide is impaired but is increased when the degradation of ceramide is blocked. Herein, we review the mechanisms of resistance to chemotherapeutic agents in terms of ceramide metabolism.

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ACCESSION NUMBER: 2000041282 EMBASE
TITLE: Systemic therapy for renal cell carcinoma.
AUTHOR: Motzer, Robert J. (correspondence); Russo, Paul
CORPORATE SOURCE: Department of Medicine, Mem. Sloan-Kettering Cancer Center, Cornell University Medical College, New York, NY, United States.
AUTHOR: Motzer, Robert J. (correspondence)
CORPORATE SOURCE: Department of Medicine, Memorial Sloan-Kettering Can. Center, Cornell University Medical College, New York, NY, United States.

SOURCE: Journal of Urology, (Feb 2000) Vol. 163, No. 2,
pp. 408-417.
Refs: 176
ISSN: 0022-5347 CODEN: JOURAA
COUNTRY: United States
DOCUMENT TYPE: Journal; General Review; (Review)
FILE SEGMENT: 028 Urology and Nephrology
037 Drug Literature Index
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 10 Feb 2000
Last Updated on STN: 10 Feb 2000

AB Purpose: We review the status of systemic therapy for patients with advanced renal cell carcinoma. Materials and Methods: A literature search was performed on MEDLINE and CANCERLIT to identify results of systemic therapy for patients with renal cell carcinoma published from January 1990 through December 1998. Treatment results of chemotherapy agents, immunotherapy, combination programs and adjuvant therapy were reviewed. Results: No chemotherapy agent has produced response rates that justify its use as a single agent. Interferon- α and interleukin (IL)-2 demonstrated low response rates ranging from 10% to 20%. The results of 2 randomized trials suggest that treatment with interferon- α compared to vinblastine or medroxyprogesterone achieves a small improvement in survival. Response rates in patients treated with low dose IL-2 are similar to those achieved with a high dose bolus schedule but whether the responses are as durable is being addressed in an ongoing randomized trial. A randomized trial of interferon- α plus IL-2 compared to monotherapy with either agent showed increased toxicity but no improvement in survival. In 3 randomized trials no survival benefit was associated with adjuvant interferon- α therapy following complete resection of locally advanced renal cell carcinoma. Conclusions: Despite extensive evaluation of many different treatment modalities, metastatic renal cell carcinoma remains highly resistant to systemic therapy. A few patients exhibit complete or partial responses to interferon and/or IL-2 but most do not respond, and there are few long-term survivors. Preclinical research, and clinical evaluation of new agents and treatment programs to identify improved antitumor activity against metastases remain the highest priorities in this refractory disease.

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ACCESSION NUMBER: 1999256002 EMBASE
TITLE: Progressive disease rate as a surrogate endpoint of phase II trials for non-small-cell lung cancer.
AUTHOR: Sekine, I., Dr. (correspondence); Tamura, T.; Kunitoh, H.; Kubota, K.; Shinkai, T.; Kamiya, Y.; Saijo, N.
CORPORATE SOURCE: Int. Med. and Thorac. Oncol. Div., National Cancer Center Hospital, Tokyo, Japan. isekine@gan2.ncc.go.jp
AUTHOR: Sekine, I., Dr. (correspondence)
CORPORATE SOURCE: Int. Med. and Thorac. Oncol. Div., National Cancer Center Hospital, Tsukiji 5-1-1, Chuo-ku, Tokyo 104-0045, Japan. isekine@gan2.ncc.go.jp
AUTHOR: Sekine, I., Dr. (correspondence)
CORPORATE SOURCE: Internal Med./Thoracic Oncology Div., National Cancer Center Hospital, Tsukiji 5-1-1, Chuo-ku, Tokyo 104-0045, Japan. isekine@gan2.ncc.go.jp
SOURCE: Annals of Oncology, (1999) Vol. 10, No. 6, pp. 731-733.
Refs: 7
ISSN: 0923-7534 CODEN: ANONE2
COUNTRY: Netherlands

DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 016 Cancer
037 Drug Literature Index
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 5 Aug 1999
Last Updated on STN: 5 Aug 1999

AB Background: Although the potential activity of anticancer agents has been traditionally assessed by the response rate (RR) in phase II trials, there is an increasing need to identify alternative endpoints to evaluate the efficacy of novel types of antineoplastic agents such as cytostatic agents. However, none of the proposed alternatives have been validated. Design: RR, rate of progressive disease (PD), and median survival time (MST) were obtained from 44 treatment arms in 42 single-agent phase II trials for non-small-cell lung cancer (NSCLC). Correlations between these parameters and their significance in selection of promising drugs were evaluated. Results: The median (range) RR and PD rate per treatment arm were 17% (0%-40%) and 41% (8%-93%), respectively. The PD rate correlated more closely with MST (correlation coefficient (r) = 0.80, $P < 0.001$) than did the RR (r = 0.62, $P < 0.001$). The RR of active agents against NSCLC ranged broadly from 7% to 40%, whereas their PD rates were all 50% or less. In addition, all treatment arms with a PD rate over 50% had a poor MST of six months or shorter. Conclusions: The PD rate was potentially as good an endpoint as RR, and it may be a good candidate for the primary endpoint of phase II trials for novel types of anticancer agents.

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ACCESSION NUMBER: 1999173684 EMBASE
TITLE: New chemotherapy options for the treatment of malignant gliomas.
AUTHOR: Burton, Eric, Dr. (correspondence)
CORPORATE SOURCE: Department of Neurosurgery, M787, San Francisco, CA 94143-0112, United States.
AUTHOR: Prados, Michael
SOURCE: Current Opinion in Oncology, (1999) Vol. 11, No. 3, pp. 157-161.
Refs: 24
ISSN: 1040-8746 CODEN: CUOOE8
COUNTRY: United States
DOCUMENT TYPE: Journal; General Review; (Review)
FILE SEGMENT: 016 Cancer
030 Clinical and Experimental Pharmacology
037 Drug Literature Index
008 Neurology and Neurosurgery
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 3 Jun 1999
Last Updated on STN: 3 Jun 1999

AB Chemotherapy remains part of the treatment triad that includes surgery and radiation therapy for the management of malignant gliomas. In recent years there has been an increased understanding of the molecular pathways of malignant transformation. Based on this research, new drugs have been evaluated, with specific cellular targets in mind that can be modified or inhibited. Many of these agents are now being tested in phase I and II clinical trials and have shown some promising results. Clearly, not all patients with malignant gliomas respond equally to chemotherapy. Recent evidence suggests that certain molecular markers may predict chemosensitivity in some tumor types, particularly anaplastic oligodendroglioma. This article reviews recent trends in the use of chemotherapy and clinical trials of new therapies for adults with

malignant gliomas.

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ACCESSION NUMBER: 1999173184 EMBASE
TITLE: Prostate-specific antigen and other markers of therapeutic response.
AUTHOR: Carducci, M.A., Dr. (correspondence); DeWeese, T.L.; Nelson, J.B.
CORPORATE SOURCE: Johns Hopkins Oncology Center, 720 Rutland Avenue, Baltimore, MD 21205, United States.
SOURCE: Urologic Clinics of North America, (1999) Vol. 26, No. 2, pp. 291-302.
Refs: 83
ISSN: 0094-0143 CODEN: UCNADW
COUNTRY: United States
DOCUMENT TYPE: Journal; General Review; (Review)
FILE SEGMENT: 016 Cancer
037 Drug Literature Index
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 10 Jun 1999
Last Updated on STN: 10 Jun 1999

AB Several new agents and combinations demonstrate significant activity in the treatment of patients with hormone refractory prostate cancer . Prostate- specific antigen (PSA) is being used increasingly as the key marker of a therapeutic response in trials of new agents. This article reviews data that support this marker as a surrogate endpoint, and it discusses the issues around the appropriateness of PSA as an intermediate marker with evolving noncytotoxic agents. Other biomarkers of prostate cancer progression are not uniformly elevated in men with advanced disease; to date, they are of limited clinical use. This article also discusses the rationale and results of novel and alternative biomarkers of prostate cancer progression.

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ACCESSION NUMBER: 1999163346 EMBASE
TITLE: Novel molecular targets for prostate cancer therapy.
AUTHOR: Kamradt, J.M.; Pienta, K.J., Dr. (correspondence)
CORPORATE SOURCE: Department of Internal Medicine, Michigan Univ. Compreh. Cancer Ctr., 7303 CCGC, 1500 E Medical Center Drive, Ann Arbor, MI 48109-0946, United States.
SOURCE: Seminars in Oncology, (1999) Vol. 26, No. 2, pp. 234-243.
Refs: 97
ISSN: 0093-7754 CODEN: SOLGAV
COUNTRY: United States
DOCUMENT TYPE: Journal; General Review; (Review)
FILE SEGMENT: 016 Cancer
028 Urology and Nephrology
030 Clinical and Experimental Pharmacology
037 Drug Literature Index
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 27 May 1999
Last Updated on STN: 27 May 1999

AB The treatment options available for advanced prostate cancer are increasing. These improved therapies are the result of research involving cellular targets other than DNA proliferation. For example, therapy directed against the intracellular matrix has yielded clinical

responses in patients. Other novel targets are being investigated. This review examines both laboratory and clinical advances using cell structure, growth factors, differentiating agents, angiogenesis, metastasis, and the cell cycle in the treatment of prostate cancer

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ACCESSION NUMBER: 1997207136 EMBASE

TITLE: Prostate cancer.

AUTHOR: Small, Eric J., Dr. (correspondence)

CORPORATE SOURCE: Univ. of California, San Francisco, Mount Zion Cancer Center, San Francisco, CA 94115, United States.

AUTHOR: Small, Eric J., Dr. (correspondence)

CORPORATE SOURCE: University of California, Mount Zion Cancer Center, San Francisco, CA 94115, United States.

SOURCE: Current Opinion in Oncology, (1997) Vol. 9, No. 3, pp. 277-286.

Refs: 91

ISSN: 1040-8746 CODEN: CUOOE8

COUNTRY: United States

DOCUMENT TYPE: Journal; General Review; (Review)

FILE SEGMENT: 016 Cancer
028 Urology and Nephrology
036 Health Policy, Economics and Management
037 Drug Literature Index
038 Adverse Reactions Titles
005 General Pathology and Pathological Anatomy

LANGUAGE: English

SUMMARY LANGUAGE: English

ENTRY DATE: Entered STN: 31 Jul 1997

Last Updated on STN: 31 Jul 1997

AB Prostate cancer accounted for over 41,000 deaths in the United States in 1996. Prostate-specific antigen (PSA) screening is capable of detecting prostate cancer and appears to detect cancers that are both clinically significant as well as organ-confined, and therefore potentially curable. The positive predictive value of PSA value has been increased by the use of the free-to-total PSA ratio. The early detection of a large number of nonpalpable tumors has mandated the development of new risk assessment schemas, which include nomograms and equations in which Gleason score, PSA, and clinical stage play a prominent role. Definitive answers to the question of watchful waiting versus intervention await conclusion of the prostate cancer intervention-versus-observation trial. For both radical prostatectomy and radiation therapy, one means of potentially reducing the risk of relapse is the use of androgen deprivation. Neoadjuvant androgen deprivation prior to surgery results in a lower incidence of positive surgical margins, but impact on survival is unknown. By contrast, the use of concurrent androgen deprivation appears to be associated with enhanced survival in patients treated with definitive radiotherapy. For good risk tumors, modern brachytherapy results in freedom from biochemical relapse rates similar to those observed with surgery and external beam radiation therapy. The best therapy for patients with positive margins or serologic progression, including radiation therapy, remains to be identified. The widespread availability of PSA testing has led to an empirically driven redefinition of advanced disease and includes patients with earlier stage disease in which primary treatment has failed. In these patients, debate remains as to whether combined androgen deprivation is superior to monotherapy. A comparison of flutamide with bicalutamide awaits maturation of survival data. The utility of antiandrogen withdrawal in patients with progressive disease despite androgen deprivation has been confirmed. Thereafter,

second-line hormonal maneuvers may be appropriate. In patients with truly hormone refractory prostate cancer, a variety of nonhormonal agents, including estramustine-based therapy, suramin, mitoxantrone, and doxorubicin-based regimens have demonstrated activity and remain as options.

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ACCESSION NUMBER: 1997162008 EMBASE
TITLE: Tumor angiogenesis: Tutorial on implications for imaging.
AUTHOR: Passe, Theodore J., Dr. (correspondence); Bluemke, David A.; Siegelman, Stanley S.
CORPORATE SOURCE: Russell H. Morgan Dept. of Radiology, Johns Hopkins Med. Institutions, 600 N Wolfe St, Baltimore, MD 21287, United States.
AUTHOR: Passe, Theodore J., Dr. (correspondence)
CORPORATE SOURCE: Russel H. Morgan Dept. of Radiology, Johns Hopkins Medical Institutions, 600 N Wolfe St, Baltimore, MD 21287, United States.
SOURCE: Radiology, (Jun 1997) Vol. 203, No. 3, pp. 593-600.
Refs: 82
ISSN: 0033-8419 CODEN: RADLAX
COUNTRY: United States
DOCUMENT TYPE: Journal; General Review; (Review)
FILE SEGMENT: 014 Radiology
016 Cancer
037 Drug Literature Index
LANGUAGE: English
ENTRY DATE: Entered STN: 18 Jun 1997
Last Updated on STN: 18 Jun 1997

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ACCESSION NUMBER: 1997144696 EMBASE
TITLE: Malignant brain tumors in the elderly.
AUTHOR: Fernandez, P.M.; Brem, S., Dr. (correspondence)
CORPORATE SOURCE: HLM Cancer Center/Research Institute, 12902 Magnolia Drive, Tampa, FL 33612-9197, United States.
SOURCE: Clinics in Geriatric Medicine, (1997) Vol. 13, No. 2, pp. 327-338.
Refs: 56
ISSN: 0749-0690 CODEN: CGMEE6
COUNTRY: United States
DOCUMENT TYPE: Journal; General Review; (Review)
FILE SEGMENT: 016 Cancer
020 Gerontology and Geriatrics
037 Drug Literature Index
038 Adverse Reactions Titles
008 Neurology and Neurosurgery
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 12 Jun 1997
Last Updated on STN: 12 Jun 1997

AB Primary malignant brain tumors are of significant concern in the elderly. The 23% increase of the average annual age-specific incidence during the last decade is a well-documented fact. The classical clinical presentation includes the signs and symptoms due to intracranial hypertension and focal symptoms that will depend on the location of the tumor. The surgical resection remains the mainstay of the management of malignant gliomas, and the extent of resection correlates

with the length of survival. Surgical decompression decreases the intracranial pressure, may improve the neurological function, increases the susceptibility of the remaining tumor cells to other treatment modalities, and provides adequate sampling for tissue diagnosis. After surgery, radiation is recognized as a very effective therapy for malignant astrocytomas. The reported median survival is 17 weeks in patients treated with surgery alone and 37 weeks in patients receiving radiation after surgery. Interstitial brachytherapy and radiosurgery allow delivery of high doses of radiation to the periphery of the tumor while sparing the normal brain. They have become an important alternative for selected patients. The nitrosoureas, procarbazine and vincristine, are agents commonly used for brain tumors. Clinical and experimental data indicate that older patients are less likely to respond to chemotherapy agents. Novel delivery systems using biodegradable polymers with slow release of BCNU prolong survival for patients with recurrent malignant glioma. New investigational drugs such as 9-AC, CI-980, suramin, and RMP-7 are being evaluated in current clinical trials to treat both newly diagnosed and recurrent brain tumors.

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ACCESSION NUMBER: 1997118709 EMBASE
TITLE: The use of prostate-specific antigen as a surrogate end point in the treatment of patients with hormone refractory prostate cancer.
AUTHOR: Smith, D.C.; Pienta, K.J., Dr. (correspondence)
CORPORATE SOURCE: 5510 MSRB I, 1150 West Medical Center Drive, Ann Arbor, MI 48109-0680, United States.
SOURCE: Urologic Clinics of North America, (1997) Vol. 24, No. 2, pp. 433-437.
Refs: 12
ISSN: 0094-0143 CODEN: UCNADW
COUNTRY: United States
DOCUMENT TYPE: Journal; General Review; (Review)
FILE SEGMENT: 016 Cancer
028 Urology and Nephrology
030 Clinical and Experimental Pharmacology
037 Drug Literature Index
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 20 May 1997
Last Updated on STN: 20 May 1997

AB Prostate-specific antigen increasingly is being used as a surrogate end point in trials of new agents in patients with hormone refractory prostate cancer. This article reviews data that support this marker as a surrogate end point and the contradictory data reported recently for trials of suramin. These contrasting views may originate in the different mechanisms of actions of the agents studied. These data suggest that a decline in prostate-specific antigen of at least 50% from baseline may be an important predictor of survival for patients receiving cytotoxic therapy.

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ACCESSION NUMBER: 1996313737 EMBASE
TITLE: Hormone refractory prostate cancer.
AUTHOR: Sternberg, Cora N., Dr. (correspondence); Lanari, Alessandra
CORPORATE SOURCE: Department of Medical Oncology, San Raffaele Scientific Institute, Rome, Italy.
SOURCE: Current Opinion in Urology, (1996) Vol. 6, No. 5,

pp. 258-263.
Refs: 54
ISSN: 0963-0643 CODEN: CUOUEQ
COUNTRY: United Kingdom
DOCUMENT TYPE: Journal; (Short Survey)
FILE SEGMENT: 016 Cancer
028 Urology and Nephrology
037 Drug Literature Index
038 Adverse Reactions Titles
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 6 Nov 1996
Last Updated on STN: 6 Nov 1996

AB Hormone refractory prostate cancer remains a challenge, but it is not as resistant to treatment as previously believed. After failure of initial hormone therapy, various treatment options are available that may provide objective remission and palliate the symptoms of disease. Advances in molecular biology together with an increased understanding of the biology of the disease will direct future therapeutic strategies.

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ACCESSION NUMBER: 1996130833 EMBASE
TITLE: Comparison of several antiangiogenic regimens alone and with cytotoxic therapies in the Lewis lung carcinoma.
AUTHOR: Teicher, Beverly A. (correspondence); Holden, Sylvia A.; Ara, Gulshan; Korb, Timothy; Menon, Krishna
CORPORATE SOURCE: Dana-Farber Cancer Institute, 44 Binney Street, Boston, MA 02115, United States.
SOURCE: Cancer Chemotherapy and Pharmacology, (1996) Vol. 38, No. 2, pp. 169-177.
Refs: 122
ISSN: 0344-5704 CODEN: CCPHDZ
COUNTRY: Germany
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 015 Chest Diseases, Thoracic Surgery and Tuberculosis
016 Cancer
030 Clinical and Experimental Pharmacology
037 Drug Literature Index
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 29 May 1996
Last Updated on STN: 29 May 1996

AB The efficacy of several potential antiangiogenic agents, TNP-470, minocycline, suramin, genistein, interferon δ 4, 14(sulfated)- β -cyclodextrin and tetrahydrocortisol, alone and in combination with cytotoxic therapies was examined against primary and metastatic Lewis lung carcinoma. The antiangiogenic agents when administered as single agents or in two-agent combinations were only modestly active as antitumor agents. Three antiangiogenic agent combinations, TNP470/minocycline, TNP-470/14(SO4) β CD/THC and minocycline/14(SO4) β CD/THC, produced significant increases in tumor growth delay and decreases in the number of lung metastases when administered along with cyclophosphamide compared with cyclophosphamide alone. Two antiangiogenic agent combinations, minocycline/interferon δ 4 and minocycline/14 (SO4) β CD/THC, produced significant decreases in the number of lung metastases when administered alone with adriamycin compared with adriamycin alone. The antiangiogenic combinations of TNP-470/minocycline, TNP-470/suramin, TNP-470/genistein, TNP-470/interferon δ 4 and TNP-470/14(SO4) β CD/THC, resulted in increased tumor

growth delays when administered along with CDDP, BCNU, fractionated radiation or 5-fluorouracil. There was not always a direct correlation between the antiangiogenic regimen that was most beneficial against the primary tumor as compared with disease metastatic to the lungs. These studies establish that a broad range of antiangiogenic therapies can interact in a positive manner with cytotoxic therapies.

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ACCESSION NUMBER: 1994367179 EMBASE
TITLE: The management of patients with advanced germ cell tumors: Seminoma and nonseminoma.
AUTHOR: Law, T.M.; Motzer, R.J., Dr. (correspondence); Bajorin, D.F.; Bosl, G.J.
CORPORATE SOURCE: Memorial Hospital, 1275 York Avenue, New York, NY 10021, United States.
SOURCE: Urologic Clinics of North America, (1994) Vol. 21, No. 4, pp. 773-783.
ISSN: 0094-0143 CODEN: UCNADW
COUNTRY: United States
DOCUMENT TYPE: Journal; General Review; (Review)
FILE SEGMENT: 016 Cancer
028 Urology and Nephrology
037 Drug Literature Index
038 Adverse Reactions Titles
009 Surgery
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 5 Jan 1995
Last Updated on STN: 5 Jan 1995

AB Risk stratification of GCT patients to good- and poor-risk therapies is included in the standard management of patients with advanced GCT. Therapy for good-risk GCT patients at our center consists of four cycles of EP. It is of paramount importance that patients with poor-risk GCT be referred to centers for participation in clinical trials with the intent of increasing the proportion of patients who are cured. For patients who do not achieve a durable CR to first-line chemotherapy, cisplatin plus ifosfamide (VIP, VeIP) and high-dose carboplatin and etoposide with AUBMT are effective and result in the cure of some patients. The choice of which treatment is most likely to be beneficial may be directed by prognostic factors. Paclitaxel has recently been shown to have antitumor activity in GCT and will be studied in combination chemotherapy regimens. A better understanding of tumor biology may enhance our ability to stratify patients into risk groups and predict earlier those patients who are cisplatin-resistant.

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ACCESSION NUMBER: 1994181173 EMBASE
TITLE: Cancer chemotherapy and infusional scheduling.
AUTHOR: Anderson, N.; Lokich, J.J., Dr. (correspondence)
CORPORATE SOURCE: Cancer Center, 125 Parker Hill Avenue, Boston, MA 02120, United States.
SOURCE: ONCOLOGY, (1994) Vol. 8, No. 5, pp. 99-111.
ISSN: 0890-9091 CODEN: OCLGE9
COUNTRY: United States
DOCUMENT TYPE: Journal; General Review; (Review)
FILE SEGMENT: 016 Cancer
030 Clinical and Experimental Pharmacology
037 Drug Literature Index

038 Adverse Reactions Titles

LANGUAGE: English

SUMMARY LANGUAGE: English

ENTRY DATE: Entered STN: 13 Jul 1994

Last Updated on STN: 13 Jul 1994

AB The practice of infusional cancer chemotherapy has evolved over the past decade as our increased understanding for tumor cell kinetics and drug pharmacology has brought into focus the concentration x time formulation and its importance in tumor cell killing and host tolerance. Technologic advances have contributed substantially to the practical capability of infusional drug delivery, with improved vascular access and ambulatory infusion pumps. In the past 10 years, infusional schedules have been used for virtually every class of antineoplastic agent and have demonstrated an improved therapeutic index by reduced or altered toxicity (doxorubicin, fluorouracil, ifosfamide, platinum analogs) or increased tumor cell killing (fluorouracil, etoposide, cladribine). Although there are few phase III trials comparing infusion and bolus administration, the evidence is clear that toxicity is altered and therapeutic benefit is not diminished by infusional schedules of drug administration.

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ACCESSION NUMBER: 1994048515 EMBASE

TITLE: Pharmacodynamic-pharmacokinetic relationships and therapeutic drug monitoring.

AUTHOR: Kobayashi, K. (correspondence); Jodrell, D.I.; Ratain, M.J.

CORPORATE SOURCE: Section of Hematology/Oncology, Department of Medicine, University of Chicago, 5841 S Maryland Avenue, Chicago, IL 60637, United States.

SOURCE: Cancer Surveys, (1993) Vol. 17, pp. 51-78.

ISSN: 0261-2429 CODEN: CASUD7

COUNTRY: United States

DOCUMENT TYPE: Journal; General Review; (Review)

FILE SEGMENT: 016 Cancer

030 Clinical and Experimental Pharmacology

037 Drug Literature Index

038 Adverse Reactions Titles

LANGUAGE: English

SUMMARY LANGUAGE: English

ENTRY DATE: Entered STN: 13 Mar 1994

Last Updated on STN: 13 Mar 1994

AB Pharmacokinetic-pharmacodynamic studies are becoming increasingly important in the development of new anti-cancer drugs. The Hill maximal effect model describes a sigmoidal dose-response relationship and has been applied to analyses of both haematological and non-haematological toxicity. This review discusses several approaches to population pharmacodynamics, including the two stage, NONMEM, and non-parametric approaches. Pharmacodynamic models for the haematological toxicity of amonafide, carboplatin, doxorubicin, etoposide, HMBA and menogaril are discussed, as are models for non-haematological toxicity. Adaptive control methods and therapeutic drug monitoring are useful in dosing drugs with narrow therapeutic windows, but the indications for using such strategies should be carefully selected. Models for 5FU, HMBA, methotrexate, 6-mercaptopurine, carboplatin and etoposide are discussed. Limited sampling strategies can facilitate the completion of pharmacokinetic studies and should be developed during phase I testing of new compounds. A new area of future importance is the investigation of drugs with active metabolites, such as the anthracyclines and amonafide.

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ACCESSION NUMBER: 1994029462 EMBASE
TITLE: Contemporary state of development of new anti-tumourous chemotherapeutic agents.
AUTHOR: Klener, P., Prof. Dr. (correspondence)
CORPORATE SOURCE: I Interni Klinika, I Lekarska Fakulta, Univerzita Karlova, U nemocnice 2, 128 08 Praha 2, Czech Republic.
SOURCE: Casopis Lekarů Ceskych, (1994) Vol. 133, No. 1, pp. 6-9.
ISSN: 0008-7335 CODEN: CLCEAL
COUNTRY: Czech Republic
DOCUMENT TYPE: Journal; (Short Survey)
FILE SEGMENT: 016 Cancer
030 Clinical and Experimental Pharmacology
037 Drug Literature Index
LANGUAGE: Czech
SUMMARY LANGUAGE: English; Czech
ENTRY DATE: Entered STN: 27 Feb 1994
Last Updated on STN: 27 Feb 1994

AB Since the end of the seventies in the development of anti-tumourous chemotherapeutic agents a certain stagnation can be observed. Only few new cytostatics were introduced into clinical practice. Although the last few years were not characterized by a basically new drug with an anti-tumourous effect, the range of cytostatics used was enriched by a major number of new cytostatics. Most of them are derivatives of drugs which proved useful in practice and which were prepared in order to enhance the anti-tumourous effect or to reduce the toxicity. The author summarizes information on these new cytostatics and initial clinical experience assembled with these drugs.

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ACCESSION NUMBER: 1992150771 EMBASE
TITLE: Chemotherapy.
AUTHOR: Kozłowski, J.M. (correspondence)
CORPORATE SOURCE: Department of Urology, Northwestern University, School of Medicine, Chicago, IL, United States.
SOURCE: Journal of Urology, (1992) Vol. 147, No. 3 II, pp. 938-941.
ISSN: 0022-5347 CODEN: JOURAA
COUNTRY: United States
DOCUMENT TYPE: Journal; Editorial
FILE SEGMENT: 016 Cancer
028 Urology and Nephrology
030 Clinical and Experimental Pharmacology
037 Drug Literature Index
038 Adverse Reactions Titles
LANGUAGE: English
ENTRY DATE: Entered STN: 7 Jun 1992
Last Updated on STN: 7 Jun 1992

L21 ANSWER 32 OF 32 BIOSIS COPYRIGHT (c) 2009 The Thomson Corporation on STN

ACCESSION NUMBER: 2002:22096 BIOSIS
DOCUMENT NUMBER: PREV200200022096
TITLE: Suramin enhances the antitumor activity of paclitaxel in mice bearing lung metastasis of human prostate tumors.
AUTHOR(S): Walsh, Colin Thomas [Reprint author]; Song, SaeHeum [Reprint author]; Wientjes, M. Guili [Reprint author]; Au, Jessie L.-S. [Reprint author]
CORPORATE SOURCE: Ohio State University, Columbus, OH, USA

SOURCE: Proceedings of the American Association for Cancer Research
Annual Meeting, (March, 2001) Vol. 42, pp. 815.
print.
Meeting Info.: 92nd Annual Meeting of the American
Association for Cancer Research. New Orleans, LA, USA.
March 24-28, 2001.
ISSN: 0197-016X.

DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)

LANGUAGE: English

ENTRY DATE: Entered STN: 26 Dec 2001
Last Updated on STN: 25 Feb 2002

=> d his

(FILE 'HOME' ENTERED AT 14:53:10 ON 21 OCT 2009)

FILE 'REGISTRY' ENTERED AT 14:53:23 ON 21 OCT 2009

L1 13 S SURAMIN

FILE 'CAPLUS' ENTERED AT 14:53:45 ON 21 OCT 2009

L2 2137 S L1

L3 629 S L2 AND (?CANCER? OR ?TUMOR? OR ?TUMOUR? OR ?NEOPLASM?)

L4 205 S L3 AND (CYTOTOXIC? OR CHEMOTHERA?)

L5 66 S L4 AND (?POTENTIAT? OR ?ENHANC? OR ?INCREAS?)

L6 11 S L5 AND (KIT OR COMPOSITION)

FILE 'REGISTRY' ENTERED AT 14:56:51 ON 21 OCT 2009

L7 1 S SURAMIN/CN

FILE 'CAPLUS' ENTERED AT 14:57:15 ON 21 OCT 2009

L8 1782 S L7

L9 605 S L8 AND (?CANCER? OR ?TUMOR? OR ?TUMOUR? OR ?NEOPLASM?)

L10 197 S L9 AND (CYTOTOXIC? OR CHEMOTHERA?)

L11 21 S L10 AND AD<20010924

L12 2 S L11 AND KIT

FILE 'MEDLINE, EMBASE, BIOSIS' ENTERED AT 15:00:22 ON 21 OCT 2009

L13 9817 S L7

FILE 'REGISTRY' ENTERED AT 15:00:35 ON 21 OCT 2009

SET SMARTSELECT ON

L14 SEL L7 1- CHEM : 9 TERMS

SET SMARTSELECT OFF

FILE 'MEDLINE, EMBASE, BIOSIS' ENTERED AT 15:00:36 ON 21 OCT 2009

L15 13200 S L14

L16 9817 S L13 AND L15

L17 6551 DUP REM L16 (3266 DUPLICATES REMOVED)

L18 1752 S L17 AND (?CANCER? OR ?TUMOR? OR ?TUMOUR? OR ?NEOPLASM?)

L19 1247 S L18 AND PD<20010924

L20 298 S L19 AND (?POTENTIAT? OR ?ENHANC? OR ?INCREAS?)

L21 32 S L20 AND (CARBOPLATIN OR PACLITAXEL OR DOCETAXEL OR GEMCITABIN

L22 0 S L21 AND (KIT OR COMPOSITION)

=>

---Logging off of STN---

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